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ATLAS
OF
CLINICAL SURGERY

WITH SPECIAL REFERENCE TO
DIAGNOSIS AND TREATMENT
FOR
PRACTITIONERS AND STUDENTS

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With 150 Colored Figures
From Models by F. Kolbow in the Pathoplastic Institute of Berlin.

VOL. II



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Fig. 60. Contractura aponeurosis palmaris (Dupuytren).

CONTRACTURA APONEUROSIS PALMARIS (*Dupuytren*)

(*Dupuytren's Contracture of Palmar aponeurosis*)

Plate XLVI, Fig. 60.

The palmar aponeurosis, the continuation of the palmaris longus muscle, which spreads over the palm and sends processes to the proximal phalanges of all the fingers, and is also connected with the skin, may be affected by chronic inflammation leading to connective-tissue formation and subsequent contracture. Hard nodules develop in the aponeurosis and skin, which finally become hard cords. These cord-like thickenings occur not only in the palm, but even more commonly in the processes of the aponeurosis connected with the second, third, fourth and fifth fingers. Contraction of these cords, which at the base of the phalanges are connected with the tendon sheaths, gives rise to an abnormal position of the fingers, called *Dupuytren's* contracture. This term signifies limitation of movement in the joints which may be of arthrogenous, neurogenous, myogenous, tendogenous, or dermatogenous origin.

The fourth and fifth fingers are those most often affected by *Dupuytren's* contracture, the second and third less often, and the thumb least often. The disease usually begins in the fourth or fifth finger and may spread to all the others. It is often symmetrical, affecting both hands at the same time and to the same extent. Before the commencement of contraction, nodular, fibrous thickenings can be felt in the skin, later on fibrous cords are formed, by which first the proximal phalanges, later on the middle phalanges become fixed in a position of flexion, while the terminal phalanges maintain their power

of extension. After some years the contraction becomes so severe that the finger is completely doubled on itself into the palm, and cannot be extended. There is generally some power of extension of the middle and terminal phalanges, but as this is painful it is avoided by the patient.

The affection occurs exclusively in men and was hence attributed to traumatic influence by *Dupuytren*. At any rate the affection is often found in people in whom the palm of the hand is exposed to continued pressure (in post-office clerks, as the result of stamping, persons who carry guns, carpenters, etc.). Some authorities attach little importance to the action of trauma, and the disease often occurs in gouty people. As the contraction is often symmetrical and equally developed on both sides, a central nervous origin is possible.

Differential Diagnosis. *Dupuytren's* contracture differs from contracture due to cutaneous scars, by the skin over it being intact. Fibromas of tendons or tendon sheaths (Fig. 49) form rounded swellings. In occupational contracture of the fingers, there are no hard cords in the palm, and the phalanges of all the fingers are usually equally flexed. In arthrogenous contracture the joints are obviously affected.

Treatment. Mechanical treatment and massage are incapable of arresting the progress of the disease. In severe cases operation is indicated, according to *Kocher* excision of the affected parts of the palmar aponeurosis. Those parts of the skin which show fibrous changes should also be removed, and the wound repaired by skin flaps. Massage, commenced soon after the operation, may give good functional results. Treatment by injection of thiosinamin is at present inconclusive.

Fig. 60 shows a case of *Dupuytren's* contracture of the fourth and fifth fingers in a man of fifty. The little finger is considerably contracted, and only the last phalanx can be freely extended. The fourth finger shows contracture of the first phalanx and commencing contracture of the second. Contracture is also beginning in the third finger. The affection was of several years duration, and caused so little trouble that operation was refused.

CONTRACTURA POST PANARITIUM TENDINOSUM

(*Contracture after Tendon Sheath Suppuration*)

Plate XLVII, Fig. 61.

Cutaneous contractures affect chiefly the flexor surface of the fingers and palm, and originate in the scars of operations, wounds, burns and inflammations. Tendon contracture is often associated with cutaneous contracture, especially when there is suppuration within the tendon-sheaths, so that the finger becomes stiff and fixed firmly in a contracted position. Fig. 61 shows a hard, slightly movable scar, extending from the flexor surface of the last joint of the middle finger to the center of the palm, arising from an incision for suppuration of the tendon sheath (cf. Fig. 93). The nature of the lesion, and the fact that there is no power of motion in the finger, shows that the flexor tendon is destroyed. Hence, the contracture is both dermatogenous and tendogenous, *i.e.* caused by contraction of both skin and tendon.

In cases where the tendon is partly destroyed, or very firmly connected with the hypertrophic cutaneous scar, operative treatment is not successful. After excision of the scar, contracture occurs in the new scar, in spite of extension of the finger, lengthening of the tendon, transplantation of tendon or catgut, or plastic operations. If the patient is incapacitated from work by the contracture, exarticulation of the fingers gives the most useful result, the use of the thumb being cultivated to take their place.

In cases of tendon-sheath suppuration, contracture may be prevented by making small lateral incisions in the finger. If the tendon is not destroyed by sup-



Fig. 62. Haemarthros — Compressio N. ulnaris



Fig. 61. Contractura post panaritium tendinosum.

puration, the skin contracture can then be prevented by early, active and passive movements, massage, baths, etc.

In cases of contracture limited to the skin, such as those after cuts and burns, keloid scars, superficial suppuration, etc., the prognosis is much better. The mobility of the scar over the deeper structures and the power of moving the individual phalanges, show that the tendon is not implicated. Excision of the scar, extension of the finger, in some cases lengthening of the tendon, and repair of the wound by skin flaps, in these cases restores the function of the finger. In young persons good results can be obtained by orthopedic treatment, when the scar is not very extensive, nor hypertrophic, nor of too long standing.

HAEMARTHROS COMPRESSIO N. ULNARIS

(*Hemarthrosis Compression of Ulnar Nerve, Neurogenous Contracture*)

Plate XLVII, Fig. 62.

Neurogenous contractures affect the hand and fingers, and result from injuries to the radial, ulnar and median nerves. They may be of peripheral or central nervous origin. In contractures of central origin, especially in the paralytic contractures due to anterior poliomyelitis, nerve transplantation, and shortening or transplantation of tendons may be performed. Treatment by massage, electricity and orthopedic apparatus is also useful. In contractures due to lesions of the peripheral nerves (division of nerve; pressure from badly united fracture, effusion of blood, or tumors on the nerve), exposure of the nerve, with excision of the injured part and subsequent suture is sometimes successful.

Fig. 62 shows a reflex contracture resulting from a blow on the ulnar side of the wrist joint, causing effusion of blood into the joint (hemarthrosis) which pressed on the ulnar nerve. Compression of the ulnar nerve by the joint effusion gave rise to "claw hand"—by hyperextension of the proximal phalanges and flexion of the second and third phalanges. There was slight swelling on the back of the wrist joint, chiefly on the ulnar side. Fluctuation was present. The sign of "snowball crunching" indicated the presence of blood clots, and therefore of hemarthrosis. The movements of the joints were limited and very painful. The joint was in a position of slight flexion, but could be easily extended.

The hand was fixed on a splint and recovery took place after absorption of the blood.

The diagnosis between the different kinds of neurogenous contractures, and between these and other contractures often requires an examination of the whole nervous system. Hysterical contracture of the knee and hip joints, which is common in children, disappears under an anæsthetic.

CONTRACTURA ISCHAEMICA BRACHII

(*Ischaemic contracture of the arm*)

Plate XLVIII, Fig. 63.

Myogenous contractures occur most commonly in the upper extremity, as a result of injuries and supuration in the muscles, which cause shortening of the muscles and their tendons. They also occur in diseases of the nervous system, both peripheral and central. Contracture also results from too long immobilization of a limb, the over-action of the flexor muscles causing flexion contracture of the arm, wrist and fingers.

These contractures are most marked in ischaemic muscular contracture (*Volkmann*), which is generally observed in the upper extremity of young persons. The causes of this condition include fractures (*e.g.* supra-condyloid fracture of the humerus), rupture of the intima of blood-vessels, obstruction of large vessels, exposure to cold, prolonged action of *Esmarch's* elastic bandage, and constriction by plaster of Paris bandages. A constricting bandage is sufficient to cause ischaemia in the arm.

The greater frequency of contracture in the upper extremity is explained by the fact that, owing to there being less muscle in the arm than in the thigh, the vessels are more easily compressed. Out of thirty-five cases collected by *Bardenheuer*, there was only one affecting the leg. The greater frequency of ischaemic contracture in young individuals is due to the greater compressibility of their muscles and vessels. In older persons great pressure on the vessels is liable to cause gangrene owing to arteriosclerosis; even slighter pressure may give rise to obliterative thrombosis and consequent gangrene.



Fig. 63. Contractura ischaemica.

It must be borne in mind that muscular tissue is more affected than skin and bone even by short interruption of the blood supply, because the compressed vessels are terminal branches.

The affection begins in the peripheral parts of the extremities. The fingers become blue, swollen, cold and moist, painful on movement, which can only be done passively, and flexed. In cases where the affection is due to tight bandaging, after early removal of the bandage the skin appears white, while the muscles feel as hard as a board and immobile, but recover after proper treatment. If the constricting bandage is allowed to remain, in a few hours the muscles become bloodless and undergo degeneration, having a waxy-yellow appearance as in typhus. As the result of extensive muscular atrophy, shrinking of the muscles takes place and causes contracture. The patients suffer severe pain for a long time after removal of the bandage. The skin of the fingers gradually becomes yellowish-white like parchment. The swelling of the fingers is followed by shrinking. First of all the fingers, then the metacarpal bones, and finally the wrist become fixed in a position of flexion. The fingers are eventually so strongly flexed that the hand becomes useless. The movements of the wrist are also very limited, and the muscles of the forearm become atrophied and are covered by pale skin. Sensory disorders may occur from pressure of the shrunken muscles on the nerves, and in some cases ischaemic muscular contracture is followed by ischaemic paralysis.

The clinical appearance of myogenous contracture, especially ischaemic muscular contracture, is so characteristic that it can hardly be mistaken for other forms of contracture.

Treatment. Myogenous contracture, when not of too long standing, may be improved by massage, electricity, baths and hot-air treatment. Prophecy-

HALLUX VALGUS (*Hammer-toe—Arthrogenous Contracture*)
Plate XLIX, Fig. 64.

In the foot contractures occur which are generally limited to the first and second toes. Pointed shoes cause external deviation of the great toe, known as hallux valgus. The deviation may be as much as fifty degrees, so that the great toe lies over or under the second toe. As the result of changes in the joint (atrophy, inflammation, arthritis deformans), arthrogenous contracture takes place in the metatarso-phalangeal joint, so that in advanced cases the deformity cannot be corrected. Over the projecting metatarso-phalangeal joint exostoses, clavus and bunions may develop, while an ingrowing toenail usually forms on the outer side of the great toe (Fig. 99). Clavus most commonly forms a circumscribed thickening of the horny layer of the epidermis, causing pain by pressure on the papillary nerve endings. Underneath the clavus a bursa generally forms which may suppurate (bunion) and perforate externally or into the joint. Clavus most often occurs on the first and fifth toes. In hallux valgus and in hammer-toe clavi are always found, often between two toes or under the toe-nails. Subungual exostoses also occur in these cases (Fig. 140).

Hammer-toe is an arthrogenous flexion contracture usually affecting the second toe, as the result of wearing too short boots, or secondary to hallux valgus. The first phalanx is extended, the second and third flexed. The third toe is rarely affected.

Hallux valgus and hammer-toe are often combined with flat foot, and then render walking still more awkward and painful.



Fig. 64. Hallux valgus — Hammerzehe — Arthrogene Kontraktur.

Treatment. Prophylactic treatment consists in attention to the feet, baths, cutting the toe-nails straight instead of curved, properly made boots, etc.

Hallux valgus, if it gives much trouble, is best treated by cuneiform osteotomy of the metatarsus and subsequent correction in plaster of Paris.

Hammer-toe is often treated by fixation to a splint, after correction of the deformity, but this is unsatisfactory. It is better to cut through the soft parts at the seat of flexion, and resect the joint from the extensor surface; or in bad cases to disarticulate the toe.

Exostoses can be chiseled; subungual exostoses after removal of the nail.

Clavi are best removed by the knife. In subungual clavus the nail must be removed first. Fistula from a bunion should be freely incised and cauterized; or the whole bursa may be extirpated. (For the treatment of ingrowing toe-nail and flat foot see Figs. 99 and 83).

Fig. 64 shows the result of neglect and badly fitting boots. The great toe shows typical hallux valgus. On the inner side of the metatarso-phalangeal joint is a clavus, on which opens a fistula from a bunion lying under it. On the outer side of the great toe the nail is ingrowing. The second toe is affected with hammer-toe and also clavus. The back of the foot is covered with dry eczema, due to uncleanness. Owing to these disorders and a considerable degree of flat foot the patient could hardly walk. The hallux valgus was corrected by cuneiform osteotomy of the metatarsus. The clavus and bunion were excised, and the second toe disarticulated. The eczema healed quickly with *Hebra's* ointment. After this the patient could walk normally, with a well-made boot.

RHACHITIS—INFRACTIONES CRURIS UTRIVSQUE

(*Rickets, Greenstick, Fractures of Both Legs*)

Plate L, Fig. 65.

Rickets, which is also known as the English disease, is a disturbance of growth affecting the whole skeleton. It consists in softening of the bones in the course of their growth, from defective ossification due to deficiency in calcium and magnesium phosphates. In the epiphyses there is abnormal proliferation of cartilage, and at the same time imperfect calcification of the cartilage. This causes thickening of the epiphyses and interference with the growth of the long bones in rickety children. Irregularity in the formation of the medullary spaces also plays a certain part. In the flat bones growth in thickness is hindered.

In the skull the disease affects chiefly the frontal and parietal bones. The bony substance may be so poorly developed that the bones are soft and flattened, yielding to pressure (*cranio-tabes*). In other places, especially the frontal and parietal eminences, the bones are thickened and prominent from the overformation of bony tissue. The cranial sutures and fontanelles remain open for a long time, and hydrocephalus is often present. The upper and lower maxillæ are flattened and irregularly developed, and the implantation of the teeth is irregular and abnormal.

The weight of the body causes bending of the softened bones; the spine becomes kyphotic or scoliotic; the thorax is constricted laterally, and the junctions of the cartilage and bone of the ribs become thickened (*beaded ribs* or *rickety rosary*). The pel-



Fig. 65. Rhachitis. — Infractiones cruris utriusque.

vic bones remain small, so that the rickety pelvis is a cause of obstructed labor. Lastly, in severe cases, the lower extremities become extremely bent and the bones are liable to greenstick fracture.

In the second year there is usually thickening of the epiphyses of the bones of the limbs, especially the lower ends of the ulna, radius and tibia; while the diaphyses, especially of the femur, tibia and fibula are curved. The femurs are bent outwards, the bones of the leg outwards and forwards (Fig. 65). Genu valgum occurs in the knees. The arch of the foot sinks in, causing flat foot. In severe cases of rickets the children remain so backward in growth that they become dwarfs. The so-called fetal rickets, according to recent investigations, has nothing to do with rickets. True rickets occurs exclusively in children between the first and sixth years, especially in the second year, and at puberty as late rickets, especially when heavy weights act on the limbs (genu valgum, coxa vara, scoliosis, pes valgus).

The origin of rickets and its absence in certain countries (China, Japan, Australia) is not yet quite clear, but bad hygienic conditions and especially improper feeding play an important part. Hereditary syphilis is a predisposing cause.

The disease often begins with anæmia, digestive troubles and diarrhea, while spasm of the larynx (laryngismus stridulus) or lung affections often occur and may be fatal.

Differential Diagnosis. Osteomalacia, which consists in softening of normally developed bones, occurs at a later age, more often in women. Hereditary syphilis affects fewer bones, especially the tibia, and is almost always associated with other signs of congenital syphilis—interstitial keratitis, notched teeth, etc.).

Rickety scoliosis and kyphosis are distinguished

from tuberculous spinal disease by the presence of rickety changes in other parts of the body.

The prognosis is favorable on the whole. Calcification may take place in the osseous tissue and the bones may assume a sclerotic condition, without a trace of shortening or bending being left. According to the researches of *von Schlauge* and *Veit* this occurs in the course of four years in all children who do not remain markedly backward in growth. The disease generally comes to an end about the sixth year, but it may recur afterwards, especially in the winter.

Treatment. In the first place hygienic conditions must be improved. Infants should be suckled by the mother. Later on meat, eggs and vegetables should be prescribed. Fresh air, high altitudes and sea bathing are all beneficial. Internally cod-liver oil and phosphates. The children should be kept off their feet, and sleep on hard beds. Surgical treatment consists in the treatment of green-stick fractures and in correcting the curvature of the bones of the limbs. Complicated apparatus only leads to atrophy of the limbs. Rickety spine should be treated by a strong corset.

Curvatures of the bones should only be operated on when they are severe, and then only when the disease has come to a standstill. An X-ray examination is useful; in active rickets the epiphyseal lines appear wide and irregular, sometimes with incomplete fractures and irregular arrangement of cartilage, and the cortex appears much thinned; while, in quiescent rickets, the epiphyseal lines have become regular, and the cortex appears the same thickness as the deeper parts.

As a rule, operation should not be performed before the sixth year. The curvature can be corrected manually or by the osteoclast; better still by linear or cuneiform osteotomy, followed by plaster of Paris.

Operation is also indicated in cases where there is early sclerosis of the bone, which is shown by the X-rays. In this case the curvature must be corrected by osteotomy, otherwise the bones will be arrested in growth.

Fig. 65 shows rickets affecting the whole skeleton in a girl aged four years. The left femur was so much curved and sclerosed that osteotomy was performed, while the curvature of the right femur underwent spontaneous cure. The epiphyses of the knee and ankle joints are much thickened, the upper and lower ends of the tibias are much bent. At the lower ends the X-rays showed green-stick fractures. Operation here was contra-indicated, as the X-rays showed the disease to be still in an active state. In the hip joints the X-rays showed coxa vara of the neck of the femur. The child was very feeble and backward in growth.

LUXATIO CUM FRACTURA CRURIS

(Fracture-dislocation of the leg)

PSEUDARTHROSIS

Plate LI, Fig. 66.

False joints (pseudarthrosis) occur in the leg, chiefly after oblique fractures with dislocation, or comminuted fractures; in the thigh and upper arm after transverse fractures also, as the result of interposition of the soft parts, chiefly the muscles.

Advanced age, pregnancy, rickets, syphilis, tuberculosis, may delay union of the fragments in a fracture.

Extension treatment is the best to obtain rapid and sure union. Delayed union may be accelerated by percussion of the fragments, injection of iodine and other preparations, or of blood, into the callus; by passive hyperæmia, or by the administration of phosphate of lime.

Badly united fractures can be brought into better position by the osteoclast or by osteotomy.

In the treatment of pseudarthrosis situated close to a joint resection comes into question. Pseudarthrosis in the shaft can be repaired by bone suture. When the ends of the fragments are much atrophied (X-ray examination) they must be resected before suturing. The fragments may be resected so as to overlap each other (dovetailed). The periosteum must always be spared as much as possible.

Transplantation of bone has sometimes proved successful. If no union occurs after these methods, apparatus must be worn, or amputation must be performed.



Fig. 66. Luxatio cum fractura cruris — Pseudarthrosis.

In the treatment of fractures and dislocations, especially in fracture-dislocations, the X-rays are especially useful in making an early diagnosis.

Fig. 66 shows marked deformity of the lower part of the right leg as far as the ankle joint. On the outer side there is slight outward curvature of the fibula above the external malleolus. The peripheral end of the fibula is dislocated, so that the external malleolus projects and the skin bulges on the outer side of the ankle joint. There is an outward curvature of the right tibia above the inner malleolus. The foot is in the position of advanced flat-foot.

The nature of the injury is an ununited supra-malleolar oblique fracture of the tibia. The distal part of the tibia is freely movable, although the fracture is of two years standing. X-ray examination shows that the fragments have overlapped, and that there is a united fracture of the distal end of the fibula a few centimeters above the external malleolus, in the position of the above-mentioned projection.

The patient (aged sixty), owing to effusion into the left knee and left flat foot, depended entirely on the right leg; the injury being due to the giving way of the right foot in a position of supination. The fracture dislocation had not been diagnosed, and the patient had been treated with poultices, etc. The foot was brought into proper position by resection of the lower ends of the tibia and fibula and freshening the head of the astragalus.

Nævi

NÆVUS PIGMENTOSUS PILOSUS (*Hairy Pigmentary Nævus*)
Plate LII, Fig. 67.

Nævi (or birthmarks) are congenital, fibrous new formations of the skin. Lentigines and ephelides (freckles) resemble nævi in their histological structure.

Only the larger nævi are present at birth; the rest develop during childhood and cease growing at puberty.

Nævi formed of blood-vessels are called vascular nævi (Figs. 75 and 76), while those formed of lymphatics are known as lymphangiectasis.

A special form of nævus is the pigmentary nævus. Owing to the presence of lymphatics this is called by *von Recklinghausen* lymphangio-fibroma, by *Borst* fibroma melanodes. These are often covered with hairs and are also known as pigmented hairy nævi (Fig. 67). In pigmentary nævi there is proliferation of fibrous cells in the dermis, dilatation of lymphatic vessels, and pigment within the cells of the dermis and epidermis.

These nævi are round, oval, or irregular in shape, with a sharply defined margin, and brown, yellowish-brown, blackish-brown or black color. Clinically they are divided into two forms, flat nævi, on a level with the skin, and projecting nævi.

Flat nævi occur on one side or over the whole body. Their distribution sometimes corresponds to that of the cutaneous nerves, and on this account their origin has been attributed to trophic changes



Fig. 67. Naevus pigmentosus pilosus.

in the spinal ganglia, also to fibromas of the smallest cutaneous nerves. In elephantiasis of nerves flat nævi are generally found on the body. Projecting nævi, especially pigmentary, often have a surface resembling that of a wart, and may assume a villous appearance. Lastly, papillomas, carcinomas and sarcomas may arise from nævi.

As a rule, nævi cause no trouble, but occasionally they may become ulcerated.

Differential Diagnosis. Nævi may have some resemblance to warts, fibromas and pityriasis versicolor, but the diagnosis is usually easy.

Treatment. On exposed parts of the body nævi should be excised, for cosmetic reasons. Removal is also indicated in rapidly growing nævi, and when inflammation occurs.

Ephelides may be removed by the application of strong resorcin paste.

Fig. 67 shows a very extensive pigmentary hairy nævus which was present at birth, and increased in size till the age of puberty. The borders are smooth, but the central parts of the surface are warty (nævus verrucosus). The color is blackish brown in the center and brown at the periphery.

NÆVUS NEUROMATOSUS—FIBROMA CUTIS

(*Cutaneous Fibroma*)

Plate LIII, Fig. 68.

The distribution of certain nævi in the course of nerves has been already mentioned. *Von Recklinghausen* was the first to show the connection between disseminated pigment spots and nervous diseases. The researches of *Soldan* have shown that in pigmentary nævi the presence of nerves can be demonstrated, in the sheaths of which fibromas develop which can only be seen with the microscope, but arise like the larger fibromas of nerve sheaths; also that they appear in the form of multiple soft tumors (fibroma molluscum), or as congenital elephantiasis of nerves.

Fig. 68 shows a slightly pigmented nævus extending over most of the forearm, with a bluish-red, irregular elevation in the center. The presence of numerous small, soft nodules in the skin (fibromata mollusca); also the presence of a small projecting growth, painful on pressure, which is formed by a fibroma of the nerve sheath of a large subcutaneous nerve, shows it to be a case of nævus neuromatosus. Multiple cord-like formations could be felt under the nævus, which were probably plexiform neuromas. Pigmentary spots were present over the whole body, and fibromata the size of a nut on the upper arm and axilla.

Fibromas of nerve-sheaths have been incorrectly called neuro-fibromas; but they consist of fibrous tissue only, without any proliferation of nerve fibres. They are generally multiple and disseminated over the whole body, forming small, soft fibromas when



Fig. 68. Naevus neuromatosus — Neurofibroma cutis.

they affect the fine cutaneous nerves, and are combined with numerous pigment spots (neuro-fibromatosis of *von Recklinghausen*). The small tumors may lie so closely together that the skin assumes a finely lobulated appearance (temples, neck and back). This condition has been termed elephantiasis nervorum, and consists in fibrous tissue formation with lymphatic vessels (Fig. 69). The disease is either congenital or appears at an early age, and is due to developmental disturbances. There is sometimes also a hereditary predisposition.

In distinction to these small, soft, multiple fibromas, fibromas of the larger nerve trunks appear as hard fusiform tumors of the sheaths of the cutaneous (Fig. 68) or subcutaneous nerves. They are very painful on pressure. Functional disorders occur in the form of paræsthesia.

In addition to these two forms of fibroma, there are true neuromas which resemble cirroid aneurism, and are, therefore, called cirroid neuroma or plexiform neuromas. These are formed of twisted cords which may form an inextricable network of nerve cords.

In distinction to the fibromas of nerve-sheaths, in which there is no new formation of nerve fibers, there is in true neuromas a new formation both of fibrous tissue and nerve fibers, which is due to developmental disturbance, which generally appears at birth, and chiefly affects the scalp, temples, nape of the neck and the back. In this case also there occur combinations with pigment spots, fibromatosis, fibromas of nerve-sheaths and elephantiasis of nerves.

Differential Diagnosis. Isolated fibromas of nerve-sheaths may be mistaken for other tumors, but there are generally other anomalies present, such as pigment spots, etc.

Treatment. Nævus neuromatosus should only be excised when it shows papillomatous proliferation,

or when fibromas or plexiform neuromas are situated beneath it.

Isolated fibromas of the nerve sheaths can generally be excised without injuring the nerve; but in large fibromas the nerve may have to be removed, with subsequent nerve suture. Recurrence is rare.

Multiple fibromas are apt to recur after operative interference, which seems to show that irritation and trauma favor their development. Rapidly growing tumors should be removed as they may undergo transformation into sarcoma and myxosarcoma.

Plexiform neuromas must be completely extirpated, as recurrence takes place if any part is left behind. At the same time the thickened skin should be removed, if it shows elephantiasic changes (Figs. 68 and 69). In extensive cases the operation may be done at several sittings.

Fig. 68 shows the various affections mentioned above in the left arm of a young man. The extensive *nævus pigmentosus* was present at birth. The smaller *nævus neuromatosus*, and the multiple, small, soft fibromas lying in it; the hard fibroma, arising from the sheath of a large nerve, seen at the upper end of the *nævus neuromatosus* near the bend of the elbow; also the plexiform neuroma appearing in the subcutaneous tissue in the form of twisted cords, all developed later, but had been present many years. Small pigment spots were present all over the body. There were also fibromas of different sizes in the course of the different nerves of the same arm. A fibroma situated in the axilla caused much pain, and was removed. Excision of the *nævus neuromatosus* and the underlying plexiform neuroma was performed later.



Fig. 69. Elephantiasis nervorum — Fibromata mollusca.



Fig. 70. Rhinophyma — Akne rosacea.

ELEPHANTIASIS NERVORUM (*of the Nerves*)
—**FIBROMATA MOLLUSCA**
Plate LIV, Fig. 69.

Fig. 69 shows a similar case in a girl, aged twenty. The whole of the right half of the scalp, the right side of the forehead and the ear are the seat of a lobulated growth (elephantiasis nervorum) fixed on the head like a cap. The growth was congenital, and on its surface are numerous pigment spots and soft, small, painless tumors (fibromata mollusca). Numerous cord-like formations were found in it by palpation (plexiform neuroma). The tumor was partially removed by a curved incision, the scar of which is shown in the figure. Total extirpation was performed subsequently at several sittings. Microscopic examination confirmed the above-mentioned explanation of the affection, the lymphatic vessels being increased and dilated in the region of the tumor.

ELEPHANTIASIS PENIS LYMPHANGIECTATICA

(*Lymphangiectatic elephantiasis of the penis*)

Plate LV, Fig. 71.

In distinction to congenital elephantiasis of nerves there is a second form of elephantiasis arabum, which for various reasons chiefly affects the lower extremities, and is known as acquired elephantiasis or pachydermia. It consists in a chronic, inflammatory hyperplasia, and there is no formation of true tumors. There is diffuse thickening of the connective tissue (fibromatosis), both in the cutis and in the subcutaneous tissue. Finally the muscles are attacked and replaced by hyperplastic connective tissue. The periosteum of the bones may present osteophytic deposits. Lastly, the epidermis takes part in the proliferative process, so that the skin becomes thickened and horny, or eczematous.

The affected parts thus become greatly thickened. The thickening may be uniformly distributed, or may assume a lobulated formation as in elephantiasis nervorum. In addition to the proliferation of connective tissue there is always dilatation of the blood-vessels and lymphatics. The disease thus appears to originate in lymphatic engorgement, and the proliferation of connective tissue results from lymphatic infiltration of the tissues.

All processes which give rise to lymphatic engorgement may, in certain cases, lead to elephantiasis. For this reason, in the endemic form of this elephantiasis which occurs especially in Arabia, Egypt, Australia, and generally in tropical countries, it has been assumed that the parasites (*filaria sanguinis*) block up the lymphatic vessels, causing lymphatic varices which rupture and deluge the tissues with lymph,



Fig. 71. Elephantiasis penis lymphangiectatica.

and give rise to hyperplasia of the connective tissue. The lymph vessels may be so dilated that small bladders filled with lymph may be visible on the surface of the skin.

The endemic form generally has an acute onset with fever and lymphangitis. After the acute symptoms have subsided, swelling of the lower extremities remains behind. Further attacks follow which cause increased thickening. Endemic elephantiasis principally affects the scrotum, penis, and female genitals. As in the sporadic form, the thickening is soft at first, but becomes hard later on from diffuse fibromatosis.

Sporadic elephantiasis is caused by affections which give rise to lymphatic engorgement—chronic oedema, recurrent erysipelas, chronic inflammations such as tuberculous and syphilitic, varicose ulcer, phlebitis and thrombosis of veins, and purulent inflammations (especially streptococcus infection). The lower extremities are generally affected, often in women with chronic eczema and varicose ulcer (Fig. 72). In prostitutes, the labia, clitoris and perineum sometimes become affected with elephantiasis, from gonorrheal discharges and syphilis. In men, the penis may be affected, especially after removal of the inguinal glands on both sides (Fig. 71).

In elephantiasis the tissues at first feel soft, afterwards firm and elastic. Eczema, bullae, pigmentations, scabs and crusts, condylomatous or papillomatous proliferation, or finally ulceration may occur on the surface. The leg or scrotum may be so much thickened that the patient can hardly move. Ulceration causes intolerable suffering.

Differential Diagnosis. Acquired elephantiasis differs from elephantiasis nervorum in the nature of its origin, and in the absence of true fibromas and plexiform neuromas. In partial giantism there is an overgrowth from early infancy of all the tissues, including the bones.

Treatment. As endemic elephantiasis is conveyed by means of drinking water and parasitic insects, precautionary measures must be taken for its prevention.

In sporadic elephantiasis all chronic inflammatory processes, etc., which excite the disease, must be avoided. Bubos should be incised early to avoid lymphatic obstruction, and ulcers of the foot must be treated (Fig. 72).

In slight cases of elephantiasis moderate results have been obtained by elevation of the limb, massage and injections of alcohol. More extensive cases may be treated by cuneiform excision. Ligation of the arteries of the skin is useless and dangerous. In extensive ulceration of the leg, amputation may be necessary.

Fig. 71 shows a case of acquired elephantiasis of the penis and scrotum in a man, aged forty, after extirpation of the inguinal glands on both sides. According to the patient the thickening of the penis and scrotum developed gradually during some years, and caused no inconvenience. Still greater acute swelling of the penis often developed suddenly, showing that it was a form of acquired elephantiasis which has been called lymphangiectatic. According to the patient this acute swelling subsided after a few days in bed. The thickened tissue felt soft and spongy, and appeared to consist of several lobulated growths rather than uniform thickening. The skin was pigmented and the scrotum covered with crusts, and there were numerous depressions as in rhinophyma. The patient was treated by suspension, elastic pressure, and later on cuneiform excision.



Fig. 72. Ulcus cruris varicosum Elephantiasis, Pachydermia acquisita.

ULCUS CRURIS VARICOSUM (*Varicose ulcer of the leg*)
ELEPHANTIASIS S. PACHYDERMIA ACQUISITA
(*Acquired elephantiasis or pachydermia*)
Plate LVI, Fig. 72.

In this case an elephantiasic thickening of the toes has developed in connection with a varicose ulcer of the leg; which, as already explained (Plate LV), is due to connective-tissue hyperplasia of the skin resulting from lymphatic engorgement (acquired lymphangiectatic pachydermia). The toes are enormously thickened, and constricted in places; the whole foot is also enlarged, and the arch of the foot is obliterated. The thickening of the foot continually increased, and extended to the ankle. Frequent attacks of erysipelas aggravated the affection.

At the lower third of the leg, on the inner side, is an ulcer extending over nearly the whole circumference of the leg. Ulcers develop in this situation from various causes—blows on the leg, chronic eczema, abscess, erysipelas, thrombo-phlebitis, varicose veins, burns and frost-bite.

These ulcers are most commonly connected with disturbance in the blood and lymphatic circulation both as regards their origin and chronic progress. They generally occur in old people of the poorer classes who have to do much standing, and are especially aggravated by uncleanness. They often occur on both legs. Arteriosclerosis, diabetes, and diseases of the central nervous system give rise to especially obstinate and extensive ulcers (trophic ulcer).

Varicose ulcer of the leg is characterized by its irregular slightly raised edges, while the parts round

the ulcer may be covered with scattered flabby granulations, crusts and blood-scabs (Fig. 72). There is frequent bleeding from the dilated veins at the base of the ulcer. The ulcer is often connected with a ruptured varicose vein. In small ulcers temporary healing may take place, but the scar is very thin, generally pigmented, and gives way again on the slightest cause; after which no further healing usually takes place, but the ulcer continues to extend. The whole neighborhood of the ankle joint, and even the whole leg, may be involved in ulceration, which often has a sanious discharge. In extensive ulcers there is generally severe pain and the leg becomes more or less useless owing to the extent of the ulcer and the elephantiasis.

Differential Diagnosis. Large ulcers with sanious discharge may suggest carcinoma, owing to their hard borders, but in carcinoma there are always irregular, hard-tumor masses in the whole extent of the ulcer. The possibility of transition of an ulcer of the leg to carcinoma must be borne in mind.

Gummatous ulcer is more regular, often circular, and has a punched-out appearance. The base of the ulcer is smooth and covered with a tenacious yellowish fatty core. The ulcer is generally less extensive and there is no bleeding. It heals quickly under iodide of potassium. (Fig. 123).

Treatment. To improve the circulation, rest in bed and support with elastic bandages (flannel or Japanese mull) are absolutely necessary. In cases with extensive varicose veins (Fig. 83) ligation of the saphenous vein is beneficial. The ulcer itself requires antiseptic dressings (iodoform, *Hebra's* ointment, *Lassar's* zinc paste, balsam of Peru, acetate of aluminium). The application of fenestrated compressing-bandages with *Unnas'* zinc gelatin or pep-

tonated paste is also recommended. In out-patient practice compressing bandages of mastich or starch may be used. Compressing bandages should be left on for several weeks, and the ulcer can be treated daily through the hole in the bandage.

In very obstinate ulcers incisions above the ulcer have been recommended to improve the circulation. Other measures are scraping, cauterization, or excision of the whole ulcer followed by skin grafting. Very severe cases, and those suspected of carcinoma, may require amputation.

DECOLLEMENT DE LA PEAU (*Detachment of the Skin*)

Plate LVII, Fig. 73.

Detachment of the skin is a term applied by *Morel-Lavallée* and *Köhler* to a lesion which consists in subcutaneous separation of the skin from the subjacent tissues and fascia. The skin itself is uninjured, as the lesion is produced by a force acting at a tangent which separates the skin from its foundations. The lesion is more liable to occur in the neighborhood of the elbow joint, and over the tibia (*e.g.* after being run over). Besides the detachment of skin the deeper structures may be severely injured and the bones fractured. The blood-vessels and lymphatics are injured, giving rise to effusion into the newly formed subcutaneous space and bulging of the skin. If the larger blood-vessels are torn there is subcutaneous effusion of blood and dark-red discoloration of the skin, forming an extensive tense swelling which generally disappears quickly. If the larger lymphatic vessels are torn, as usually happens, the lymphatic effusion often appears several hours after the injury. The skin is hardly altered, perhaps somewhat livid and excoriated, while the subcutaneous swelling subsides slowly, owing to the long, continual effusion of lymph.

The lymphatic effusion, which is generally more or less mixed with blood, accumulates in the dependent parts of the injured region. Fluctuation of the fluid in the subcutaneous cavity can be felt.

Treatment. Subcutaneous effusion of blood soon undergoes spontaneous absorption. The lymphatic effusion gradually disappears after repeated punc-



Fig. 73. Detachment of the Skin.

ture, injection of tincture of iodine and compression by bandages. Incision should only be performed if there is suppuration.

Fig. 73 shows a detachment of the skin resulting from a blow on the left elbow. A few days after the injury effusion took place in the subcutaneous cavity, chiefly in the forearm. The cavity was not completely filled so that several swellings are shown. There is a slight abrasion of the skin over the olecranon, the appearance and direction of which show that the blow was a tangential one. The skin is livid over the whole swelling. Yellowish fluid was evacuated by puncture, showing very slight mixture with blood.

Submucous effusion in the nasal septum and in the larynx may also be caused by the action of tangential force (generally foreign bodies). Here also the effusion only occurs where the submucous tissue is situated over a hard substratum of cartilage.

OTHAEMATOMA (*Hematoma of the Ear*)
Plate LVIII, Fig. 74.

The majority of cases of hematoma of the external ear are caused by a tangential force which tears the perichondrium from the cartilage and is followed by effusion of blood or lymph into the subcutaneous cavity. The lesion occurs especially in the upper half of the auricle, and is found in the mentally affected as the result of ill-treatment by blows on the ear, etc.; in workmen who carry loads on the shoulder which graze the ear; in carpenters through carrying planks; in butchers through carrying troughs, etc. It is also a common injury in boxers and acrobats. It generally causes little trouble.

Blood effusion is indicated by the rapid development of a tense, dark-blue swelling which, after a time, subsides. Lymph effusion is indicated by a swelling which does not develop till some time after the injury and has less tendency to subside; the skin is not discolored. Lymph effusion is nearly always slightly mixed with blood, and always forms a tense swelling, in distinction to lymph effusions in other parts. (Fig. 73).

Blood and lymph effusions in the auricle may undergo chronic inflammation, which first causes thickening, later on atrophy and necrosis of the auricle, with considerable mutilation. If the skin is much abraded, the effusion may become septic, with consequent destruction of the cartilage.

Differential Diagnosis. Cavernous heman-gioma, which often occurs in the upper part of the auricle, has some resemblance to hematoma. Hem-



Fig. 75. Haemangioma simplex.

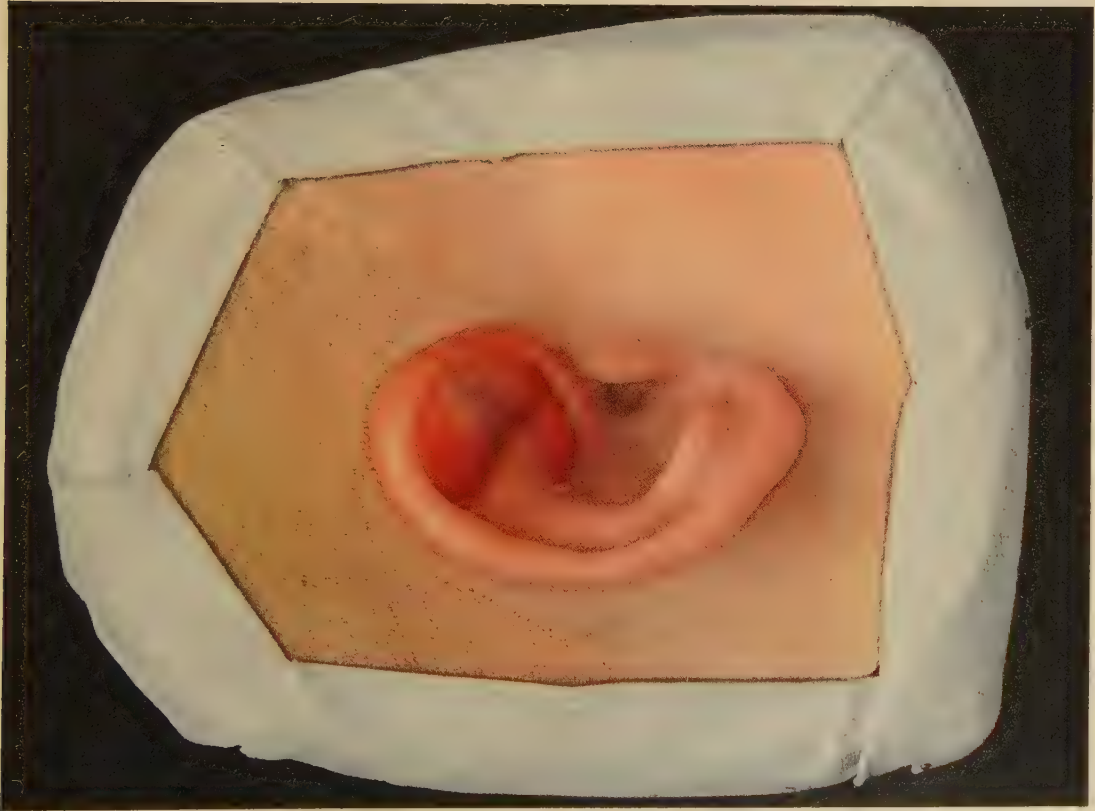


Fig. 74. Othaematoma.

angioma, however, is often congenital; it forms a tumor which can be diminished by pressure, and has a bluish coloration and an uneven surface. Other vascular anomalies are also usually present in the neighborhood of the tumor.

Treatment. Prophylactic treatment consists in the wearing of ear caps. The hematoma must be protected from injuries which may cause septic infection of the effusion. It undergoes spontaneous resolution, but more slowly than in other places. Lymph effusions recur after repeated puncture; injection of tincture of iodine and compression by strips of plaster are not of much value; massage is useful in most cases. If suppuration occurs, they must be incised.

Fig. 74 shows an effusion in the upper third of the auricle. The patient first noticed a small pimple, and as the result of scratching this the swelling gradually developed; at first soft, afterwards tense. The skin is red, not bluish red as in blood effusion. A small, blue spot in the figure represents the original pimple. The condition is one of lymph effusion. Lymph mixed with blood was evacuated by puncture, but the swelling recurred. The effusion gradually subsided after massage.

HEMANGIOMA SIMPLEX (*Simple Hemangioma*)
Plate LVIII, Fig. 75 (cf. also Figs. 76 and 81).

The term Angioma includes new growths arising from blood-vessels and lymphatics; the former are called hemangiomas, the latter lymphangiomas. Hemangiomas may be simple or cavernous (cavernoma, Figs. 36 and 80).

The red spots formed by tortuous and dilated blood-vessels (telangiectasis, *nævus vasculosus*) are by some classed as tumors and included among the simple hemangiomas; by others they are considered as hypertrophic formations, and not as true tumors, as they consist in dilatation, lengthening and tortuosity of the vessels, rather than new formation of vessels. The form known as racemose or plexiform angioma also almost always consists in a dilatation of a vascular region, not a true, new formation of vessels. It is, therefore, better to give the name cirroid aneurism to these formations, which are usually congenital and due to fetal remains, but sometimes traumatic. Lastly, neither aneurisms nor varices belong to true vascular tumors. The red spots with more or less regular outlines, often only punctiform, which occur in the skin of old persons, are also not true tumors but only dilated and tortuous blood-vessels (telangiectases). A form described by *Ziegler* as hypertrophic angioma is best named hemangio-endothelioma, as, in addition to new formation of vessels, there is extensive proliferation of the endothelium.

Clinically, we distinguish telangiectases, which are situated superficially in the skin, from simple hem-

angiomas which appear in the skin and subcutaneous tissue. The latter tumors, also called angiomas (Fig. 75), appear as raised growths with well-defined borders. The overlying skin is thin and adherent, and of a reddish-blue color. In places there are islands of normal skin. The edges of cutaneous angiomas are dark-red, slightly raised, and often bordered by an areola of fine ramifying blood-vessels. The tumors are soft, spongy, somewhat compressible, and easily movable over subjacent parts. They are sometimes present at birth and are often situated on the face, lips, cheeks and neck, in the regions of the fetal clefts. In other cases they appear soon after birth, usually in the form of slow-growing red spots. Angiomas distributed in the region of the trigeminal nerve have been called neuropathic angiomas. Angiomas may also develop in scars.

More extensive growth may form large, nodular lobulated tumors, which when situated in the orbit may be dangerous from extension to the brain; but they cannot be regarded as malignant tumors, because they give rise to no metastases.

Involution of angioma has been observed as the result of inflammation. Angiomas are usually multiple, cutaneous or subcutaneous. They may also occur in the muscles, bones, brain, breast and liver, generally in the form of cavernous hemangioma. They cause no trouble apart from that due to their disfigurement.

Differential Diagnosis. The tumors are so typical that they cannot easily be mistaken. Subcutaneous hemangioma generally appears later under the skin, which gradually assumes a bluish coloration.

Cavernous hemangioma (cf. Figs. 36 and 80) appears as a multilobular swelling, which diminishes on pressure. When it forms in the skin, the latter is colored bluish green (Fig. 81).

Treatment. Large hemangiomas of the skin and subcutaneous tissue are best excised, especially when situated on the face. Small angiomas can be treated by multiple puncture with the thermocautery into the subcutaneous fatty tissue, at several sittings (especially in subcutaneous angiomas), but the scars are often unsightly. After electrolysis the scars are smoother and less visible. Angiomas sometimes recur in the scars.

Angiomas of the eyelids, which may extend through the orbit to the brain, or those situated over a fontanelle which may implicate a sinus, also very extensive angiomas of the face are not suitable for operation. In these cases the introduction of magnesium, which causes coagulation and shrinking of the tumor, may be tried.

Fig. 75 shows a typical simple cutaneous hemangioma of the nape of the neck, which appeared as a red spot soon after birth and ceased growing after the second year. The borders of the growth are red and show small, ramifying blood-vessels. The center is bluish red and partly covered by normal skin. The tumor was soft, freely movable over subjacent parts and sharply defined. It was excised with subsequent suture.



Fig. 76. Naevus vasculosus.



Fig. 77. Haematoma diffusum — Haemophilia.

NÆVUS VASCULOSUS (*Vascular Nævus*)
Plate LIX, Fig. 76.

In distinction to the projecting hemangioma we find in telangiectasis a flat reddening in the skin which may be punctiform, annular, or of various shapes.

The greatest degree of telangiectasis is attained in the so-called vascular nævus which most often occurs on the face, and is either congenital or appears soon after birth as a red spot. This rapidly extends and often spreads irregularly over half the face. The edges are jagged and show fine ramifying vessels. The coloration of the skin varies, and there are usually different tints in the same nævus. It is often dark purple in the center and bright red at the periphery. It is often broken up by normal skin, giving a variegated appearance. Spontaneous involution has been observed in small nævi. Apart from the disfigurement they cause no trouble.

Treatment. Good results have been obtained by X-ray treatment. Cauterization with fuming nitric acid causes the nævi to disappear and leaves smooth cicatrization. (This must be used cautiously on the eyelids).

Renal hemorrhage in hemophilia may be mistaken for renal hemorrhage due to other causes (stone, tumor, tuberculosis), but the bleeding in hemophilia quickly leads to exhaustion, and gives no evidence of other changes in the kidneys.

The hemarthrosis of bleeders is so characteristic that it can hardly be mistaken. It differs from traumatic hemarthrosis in its progressive increase and slow absorption. Myeloid sarcoma extending to the joint is characterized by rapid growth and the presence of a malignant tumor (X-ray examination), and has only a similarity to hemophilia in its early stages.

In the diffuse bleeding which sometimes occurs after operations, a diagnosis of hemophilia must not be too hastily made, as this disease is quite uncommon.

Treatment. Cutaneous and subcutaneous blood effusions should be left alone; puncture is useless, and profuse bleeding often takes place from the puncture. For the same reason puncture of a joint effusion with injection of three per cent. carbolic lotion is a doubtful procedure. Compression and extension of the joint is the best treatment.

Wounds should be plugged with iodoform gauze and tightly compressed. Bleeding from the gums and nose may be treated with the thermocautery. Bleeding after tooth extraction may be averted by plugging the socket with a wedge of cork.

The most difficult cases are those in which bleeding occurs in extensive injuries, especially when there is suppuration. The application of perchloride of iron stops the bleeding for a time, but forms a scab, and after this becomes loose bleeding recurs. There is also the danger of embolism and septic infection.

It is better to use hot gelatin solution. This must be carefully sterilized before use to free it from tetanus spores, and should always be used freshly prepared, in a ten per cent. solution. Gelatin is

also useful administered internally or by subcutaneous injection. In extensive, uncontrollable bleeding affecting the extremities amputation may have to be considered; in this, all the vessels must be carefully ligatured.

In renal hemophilia nephrotomy and nephrectomy has proved successful.

During the bleeding, which often ceases spontaneously after a time, the patient's general condition must be kept up by forced nourishment. Bleeders must naturally avoid everything which may cause bleeding.

Fig. 77 shows blood effusion into the subcutaneous and subconjunctival tissue of both eyelids, and an extensive hematoma on the left side of the forehead in a child aged six years, who belonged to a family of bleeders. The effusions occurred spontaneously; the one on the forehead occurred intermittently for a time and then gradually subsided. There was no bleeding in any other part of the body.

SUGGILLATIONES ET SUFFUSIONES

(*Suggillations and Suffusions*)

HÆMATOMA SUBCUTANEUM (*Subcutaneous Hematoma*)

Plate LX, Fig. 78.

Hemorrhages into the skin when of small extent are called petechiæ or ecchymoses (Fig. 79); when of larger extent suggillations or suffusions (Fig. 79). Hemorrhages into cavities are called hematomas. The latter often occur in the subcutaneous tissue, giving rise to convex swellings of the skin (Fig. 77). Subcutaneous hematomas are common after all kinds of injury—gunshot wounds, fractures, contusions, punctured wounds, etc.; also as the result of secondary hemorrhage after operations. In these cases the skin assumes first a purple, afterwards a greenish-yellow coloration, which extends beyond the area of the hematoma and persists for several weeks. There is often a visible swelling with fluctuation. Patients complain of slight pain and a feeling of tension. If the swelling persists, the sensation of “snowball crunching,” which is characteristic of all blood effusions, is felt by palpation.

In parts where the skin is loosely attached, as in the eyelids (Fig. 77) or scrotum, there is much swelling and discoloration of the skin. After injury to a large blood-vessel, enormous, often pulsating, swellings may occur (pulsating hematoma or false aneurism).

Subcutaneous hematomas usually have ill-defined margins, owing to their gradual extent into the soft parts. Sometimes, however, they become encapsuled, and periosteal hematomas of the scalp are



Fig. 78. Sugillationes et Suffusiones — Haematoma subcutaneum.

surrounded by a wall of bony hardness (also in cephalhematoma).

Treatment. Light compression by bandages soon causes resorption of the effusion. In delayed resorption the fluid may be evacuated by puncture. If suppuration occurs an incision must be made.

Fig. 78 shows extensive suggillations and suffusions of the skin of the whole arm, which is colored purple, brownish-red, green and yellow. The presence of a subcutaneous hematoma is shown by swelling and fluctuation.

It is a typical case of gunshot injury to the soft parts, in which the apertures of entry and exit are characteristic. The aperture of entry is smaller than that of exit and shows radiating processes in the skin. The skin is colored black and contains granules of powder, owing to the shot being fired at close quarters. In shot wounds of the face these powder granules remain for a long time after the wound has healed, and cause an unsightly appearance. The aperture of exit is larger with irregular everted borders. These wounds are typical of modern projectiles with great penetrating power.

Septic infection does not usually occur in gunshot wounds, as the bactericidal power of the organism is sufficient to counteract the slight infection caused by projectiles. Even infected foreign bodies, such as shreds of cloth, may heal up in the body.

The prognosis of gunshot wounds of the soft parts is good if undue interference is avoided. All probing of the wound and search for the bullet is to be condemned, as it generally sets up virulent infection of the wound. Disinfection of the wound is also unnecessary. The best treatment is to apply an antiseptic sterilized gauze dressing (iodoform gauze if there is much bleeding) and keep the part at rest; in the extremities by the aid of plaster of Paris. By

this simple treatment, first introduced by *von Bergmann*, the best results are obtained, not only in gunshot wounds of the soft parts, but also in wounds of the joints and bones, even comminuted fractures.

In gunshot injuries of large blood-vessels operative interference is necessary; *e.g.* ligation of the middle meningeal artery.

If the wound becomes infected, as often happens after injuries with explosive bullets (dum-dum bullets, etc.), a free incision must be made to give outlet to the pus. Bullets and pieces of clothing which have become healed over may give rise to abscess after some years.

As a rule bullets should be left alone; a bullet has even remained in the apex of the heart without causing trouble (*Trendelenburg*). Only superficially situated bullets should be removed, after locating them by means of the X-rays. Bullets in the frontal or maxillary sinuses, or in the mastoid process should be removed, as they give rise to pain and chronic catarrh. Bullets should also be removed which cause pressure on tendons and nerves, or are situated in the phalanges, or prevent union of fractures.

Blank cartridges, in which tetanus spores are often present, should be removed on account of the danger of tetanus. In war, there is always a danger of tetanus infection of every large bullet wound, from the presence of tetanus bacilli in the ground on which the wounded lie. As the treatment of antitoxin is only efficacious before the tetanus appears and is too complicated to be used in warfare, the author recommends, on the strength of experimental research, the application of fat to wounds suspected of tetanus infection, as fatty substances attenuate the tetanus toxin (Surgical Congress, 1907 *Bockenheimer's* anti-tetanus ointment).

The search for deep-seated bullets in the brain causes much injury. Accumulation of blood or cerebrospinal fluid, may abolish the reflexes for a time,

and paralysis may appear, but in spite of this recovery may take place after a time.

Bullets situated outside the cortex of the brain must be removed when convulsions occur from pressure of the bullet, or a splinter of bone, or an accumulation of blood or pus.

Effusion of blood in the thorax through wound of the lung should be left to be resorbed. If it becomes so extensive as to displace the heart puncture must be performed, and if suppuration occurs resection of the ribs.

In gunshot wounds of the heart, free exposure of the organ may be performed in some cases.

Gunshot wounds of the abdomen require laparotomy at the earliest possible opportunity.

In wounds of the larynx immediate tracheotomy is necessary to avoid death from asphyxia.

In the above-mentioned cases resorption of the blood effusion and healing of the wounds takes place in a few weeks under the application of fixed aseptic dressings.

PETECHIÆ ET HÆMORRHAGIÆ PER COMPRESSIONEM

(Petechiæ and Hemorrhage from Compression)

Plate LXI, Fig. 79.

Punctiform and striate hemorrhages in the skin in the form of petechiæ and ecchymoses, and diffuse-cutaneous extravasations of blood are included in the term congestive hemorrhages. These appear in the head and neck; hemorrhage from compression of the lower parts of the body generally occurs in the thorax. Sometimes subconjunctival effusion of blood occurs after abdominal compression—an important point in criminal and accident cases in which there is no visible lesion of the abdomen. The sudden appearance of this extensive hemorrhage in the head and neck causes a dark-blue coloration of the skin, protrusion of the eyes, and a swollen and bloated appearance of the skin and mucous membranes. It occurs in cases of crush, run-over cases, and compression by machinery, and is due to back pressure on the valveless veins of the neck from compression of the thorax and abdomen, with rupture of the veins and infiltration of blood into the tissues. There is no hemorrhage into the brain or its membranes. The fundus oculi is normal, as the intra-ocular pressure prevents extravasation of blood from the retinal vessels.

The diagnosis is easy, and treatment consists only in rest in bed.

Fig. 79 shows a case of congestive hemorrhage due to compression of the thorax in a rolling mill. The whole face was colored dark purple and the mucous membranes of the lips and nostrils were swollen.



Fig. 79. Petechiae et Haemorrhagiae per compressionem.

There was also subconjunctival effusion of blood. In the neck, the continuous purple coloration of the face was replaced by a brighter red coloration in the form of stripes (petechiæ and ecchymoses). The petechiæ were situated over the shoulder and the upper part of the back; also in the auditory canal and the tympanic membrane. No visible lesion was present. The swelling of the face disappeared in a few days, and the purple coloration subsided in the course of time without any treatment. The discoloration remained longest in the eyelids and conjunctiva.

HÆMANGIOMA CAVERNOSUM SUBCUTANEUM

(*Subcutaneous Cavernous Hemangioma*)

Plate LXII, Fig. 80.

Fig. 80 shows a subcutaneous cavernous hemangioma, which often occurs in the region of the rectus abdominis muscle, sometimes in the muscle itself. Mention has already been made of cavernoma in Plate XXVII. They occur most frequently in the skin and subcutaneous tissue, where their purple color and lobulated surface has somewhat the appearance of a mulberry. They are often combined with simple hemangioma or with telangiectases, and often appear soon after birth. In cutaneous hemangioma the skin is much thinned and appears lobulated and of a bluish-black color. In subcutaneous hemangioma the skin may be unaltered at first, or slightly irregular and marked by telangiectases. Afterwards the skin becomes thinned or destroyed by pressure of the subcutaneous growth, and assumes various colors (Fig. 80). In the case represented in the figure the skin is already destroyed over the blue parts of the growth, and is of a livid color at the periphery. The growth is encapsuled and freely movable over the abdominal fascia (in distinction to infiltrating cavernoma). In some parts the cavernous spaces can be seen through the surface. In the center of the growth the skin is yellow in some parts and brown in others. The growth was soft, elastic and compressible; in some places there was thrombosis with consequent shrinking. The growth had remained stationary for a year.

Subcutaneous cavernomas of the scalp require special mention, as they may communicate with a



Fig. 80. Haemangioma cavernosum subcutaneum.

HÆMANGIOMA CUTANEUM ET SUBCUTANEUM
(*Subcutaneous and Cutaneous Hemangioma*)
TELEANGIEKTASIAE (*Teleangiectases*)
Plate LXIII, Fig. 81.

Fig. 81 shows a combination of cutaneous and subcutaneous hemangiomas with telangiectases, affecting the leg. The telangiectases are seen as red spots, in some places arranged in the form of a wreath. There is also an extensive subcutaneous hemangioma, of a bluish-red color, with more or less normally colored skin in the central parts. These growths may remain covered by intact skin for a long time, while the growth seen through it gives it a bluish coloration. In this case, at the lower part of the subcutaneous hemangioma, there were cutaneous hemangiomas in the form of more elevated, round formations in the skin, resembling the simple cutaneous hemangioma represented in Fig. 75. In the whole region of the subcutaneous hemangioma fine ramifying blood-vessels can be seen. In the face, combinations of cutaneous and subcutaneous hemangiomas sometimes form a characteristic appearance, the subcutaneous growth giving a blue color to the skin, while the cutaneous angioma appears in the form of lobulated growths or of bluish-red nodules projecting from the surface. In Fig. 81 the difference in color between the cutaneous and the subcutaneous hemangiomas is very marked, the former being red, the latter bluish in color. A combination of subcutaneous with cutaneous hemangioma and telangiectases is not very rare. The cutaneous hemangioma sometimes develops when the subcutaneous growth appears under the skin.

For **Differential Diagnosis** and **Treatment** see Fig. 75.



Fig. 81. Hämangioma cutaneum et subcutaneum — Teleangiectasiae.



Fig. 82. Aneurysma arteriale.

ANEURISMA ARTERIALE (*Arterial Aneurism*)

Plate LXIV, Fig. 82.

An aneurism is a partial dilatation of an artery. The term *true aneurism* is applied to those dilatations which are formed by all the three coats of the artery. Through wearing away of the arterial wall, the blood escapes from the vessel and is enclosed by the neighboring soft parts, forming a *false aneurism*. A form of false aneurism has already been mentioned as pulsating hematoma (Fig. 78); in this case there is a subcutaneous injury to large blood-vessels.

In both true and false aneurism we distinguish a circumscribed and a diffuse form, but the classification of aneurisms into cylindrical, saccular and fusiform is of no importance, and only has a clinical interest in cirroid aneurisms (Fig. 75).

True aneurisms are caused by disease of the arterial walls from infective diseases, chiefly syphilis. When the morbid processes extend over large areas of the arterial system the aneurisms may be multiple. These occur especially in the small arteries of the brain, sometimes also in the lungs, and by their rupture give rise to multiple apoplexy. This occurs chiefly in syphilitic disease of the arteries, and in the arteriosclerosis of young people.

True aneurisms are often situated in the ascending aorta (syphilis), also in places where the arteries are liable to traction or pressure from flexion of the extremities: *e.g.* aneurism of the femoral artery from pressure of an osseous growth ("rider's bone") in the adductor muscle; aneurism at the entrance of the femoral artery in *Hunter's* canal; popliteal aneurism, etc.

False aneurisms may arise from true aneurisms (consecutive false aneurism), or from injury to an artery, causing pulsating hematoma (traumatic false aneurism). Aneurisms only attain large dimensions when they are surrounded by soft tissues (skin, muscle, and fat). They are at first diffuse and ill-defined, but eventually become circumscribed swellings, owing to the formation of a connective-tissue capsule from the surrounding tissues.

If both artery and vein are injured, which happens in the majority of cases, an *arterio-venous aneurism* is produced. This is called *aneurismal varix* when there is direct communication between the artery and vein, and a varicose swelling of the latter; *varicose aneurism* when the two vessels communicate through a sac which is formed between them. However, this distinction is not always evident clinically, especially when a series of inextricable sacs and communications is formed through multiple perforations of the artery and vein. Traumatic aneurisms, both arterial and arterio-venous, were formerly common in the bend of the elbow as the result of phlebotomy. They generally arise from punctured wounds, or gunshot wounds with modern bullets.

Clinically, both true and false aneurisms are of gradual development, as in traumatic aneurism there is also a long interval before the sac is formed. The sac may attain the size of a man's head, forming a visibly pulsating swelling, the pulsation ceasing after compression of the artery on the side next the heart. The pulsation may be absent when the sac wall has become thickened by thrombosis. The swelling can be diminished by pressure. On auscultation of the sac a bruit is heard, which is synchronous with systole of the heart in arterial aneurism; irregular during both systole and diastole, in arterio-venous aneurism. In the latter condition there is congestion in the region of the vein, with consecutive disturbance of nutrition, eczema, ulcers, and abscess formation.

Aneurisms as a rule have a slow but persistent growth, and tend to eventual rupture. In arterial aneurism a cure sometimes occurs from thrombosis.

Aneurisms often cause severe symptoms from pressure on the neighboring organs; *e.g.* paræsthesias, neuralgia and paralysis from pressure on the nerves; congestion and elephantiasis from pressure on the veins. A large aneurism may cause atrophy of the bones from pressure (sternum and vertebræ).

Differential Diagnosis. True aneurisms can be distinguished from false traumatic aneurisms by careful examination. Abscesses, or benign and malignant tumors, especially sarcoma, when they receive pulsation from an underlying vessel, may be mistaken for aneurism. Aneurisms in which there is no pulsation or bruit, owing to thickening of their walls from thrombosis, and which have caused inflammatory changes in the skin by pressure, may be mistaken for abscesses and be incised.

In cavernoma there is dilatation of the vessels but no pulsation. Racemose aneurism presents itself as an irregular serpentine arterial swelling caused by the tortuous dilatation of a vascular area.

In many cases the X-rays are useful in the diagnosis of aneurism, which gives a dark shadow in the X-ray picture.

The prognosis of aneurism is always unfavorable.

Treatment. For large spontaneous subcutaneous aneurisms, the injection of coagulating fluids has been recommended, but these are not free from danger. The best is injection of solution of gelatin into the sac. Other methods, which also aim at coagulation, are the introduction of needles or magnesium into the sac, and electropuncture. In the extremities, digital compression or compression by instruments generally causes only temporary improvement. Compression of the common carotid artery and the internal carotid

are not without danger, as they may cause convulsions and unconsciousness.

The most certain method is ligation of the vessel above and below the sac and removal of the sac.

In arterio-venous aneurism all the sacs must be removed after ligation of all the vessels connected with them. Ligation of the common carotid, which may lead to softening of the brain, may be performed if a temporary ligature of the carotid is well borne.

The ideal method is extirpation of the aneurism with restoration of the blood-stream by suture of the vessel (*Payr*) with the aid of prothesis, which avoids such complications as softening of the brain and gangrene of the extremities after ligation of the main vessel. *Lexer* recommends lateral suture of the vessels, circular suture, or transplantation of vessels.

In some cases peripheral ligation only is possible; *e.g.* in aneurism of the subclavian artery. In the extremities, when there is much disturbance of nutrition, the question of amputation arises.

Internal medication consists in the administration of iodide of potassium, with a view to the syphilitic origin of aneurism.

Fig. 82 shows a visibly pulsating swelling in the region of the sterno-clavicular joint in a middle-aged man with a probable history of syphilis. It consists in a circumscribed arterial aneurism, and presented all the clinical symptoms of arterial aneurism—pulsation, diminution on pressure, systolic bruit and buzzing over the swelling. The swelling increased in size slowly but continually, and was shown by the X-rays to be an aneurism of the aorta. There was paralysis of the left recurrent laryngeal nerve from pressure of the dilated aortic arch, a characteristic symptom of aortic aneurism, which sometimes manifests itself by hoarseness; but, when there is compensation of the paralysis, it can only be recognized by laryngoscopic examination. An early symptom of

aortic aneurism is also the phenomenon first described by *Ollier*—pulsation of the larynx. When the larynx is pulled upwards there is a sensation of traction from below (“tracheal tugging”).

In this case, pressure on the brachial plexus caused paræsthesias in the right arm; pressure on the veins caused cyanosis of the face and neck; while the dysphagia from pressure on the esophagus, and dyspnoea, which frequently occur in such aneurisms, were absent.

Non-pulsating aneurisms may be mistaken for gumma, which is common in this situation. Aneurisms of the aorta are often unrecognized till they rupture, an event which may occur after sounding a stricture of the esophagus caused by the aneurism itself.

VARIX CIRSOIDES—PES VALGUS (*Cirroid Varix—Flat Foot*)
Plate LXV, Fig. 83.

The term *phlebectasis* is applied to dilatations and tortuosities of veins. They may occur in various parts of the body; *e.g.* in the inferior hemorrhoidal plexus of veins, as hemorrhoids; in the pampiniform plexus, as varicocele (this is more common on the left side owing to the fact that the left spermatic vein opens at right angles into the renal vein and is thereby more liable to backward pressure and congestion); more commonly in the veins of the leg (large and small saphenous veins), where they are known as varicose veins or varix.

Phlebectases appear as multiform tortuous blue cords (cirroid varix) clearly visible under the thinned skin, on the inner side of the leg in the region of the large saphenous vein. Varices of the small saphenous vein on the outer side of the leg and calf are less common.

Nodular swellings occur in places where the veins have valves.

In the upper extremity phlebectasis is less often observed, but may occur in connection with tumors of the neck and shoulder. Phlebectases on the abdomen (called *caput medusæ*) are due to obstruction of the portal circulation. Submucous varices occur in the esophagus and alimentary canal. Varicose veins also occur in the brain, especially in the Sylvian fissure.

Phlebectases in the legs are usually due to disturbance in the circulation; *e.g.* from the pressure of pelvic tumors. Phlebectases may occur on both sides

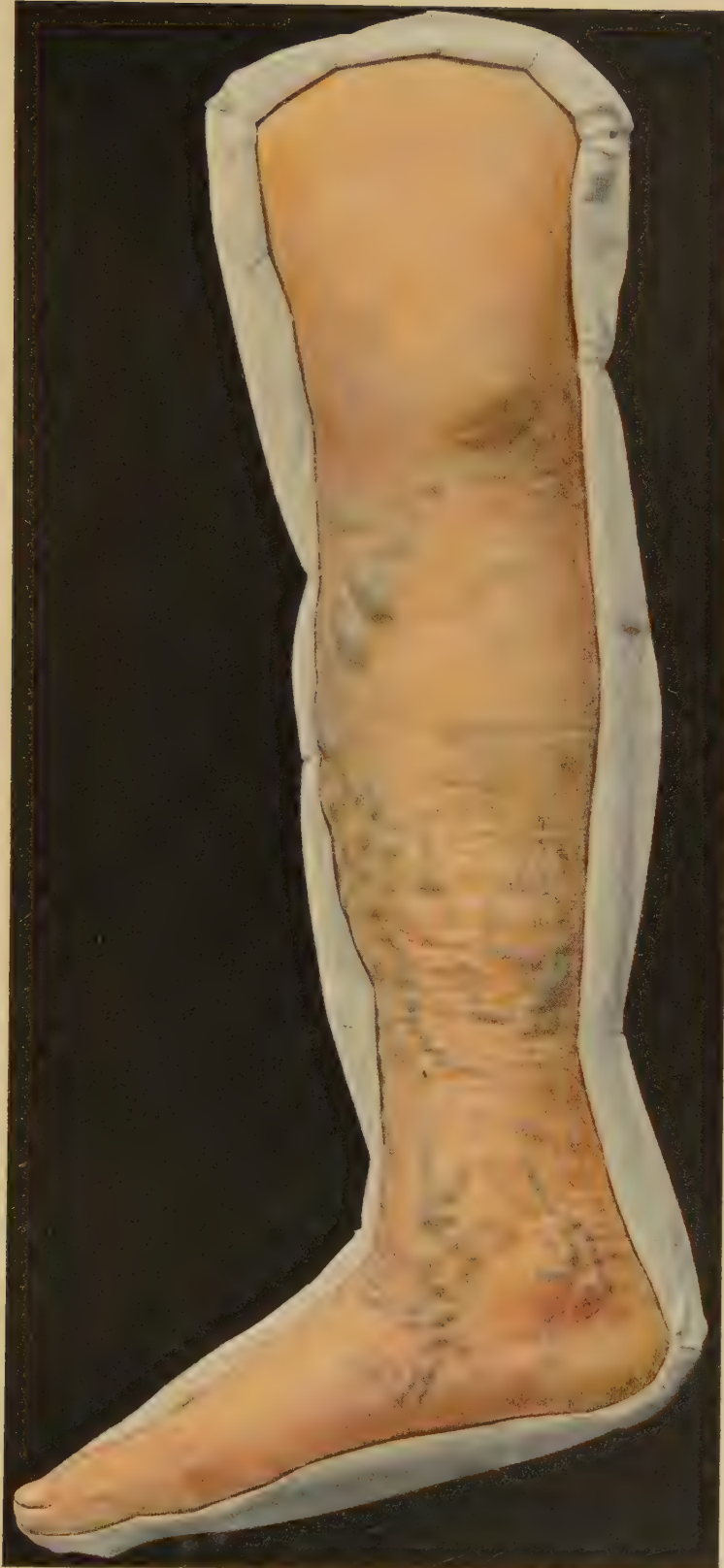


Fig. 83. Varix cirroides — Pes valgus.

and be combined with hemorrhoids, especially in women who have had many pregnancies.

Under the thinned skin hard lumps can be felt where thrombosis has occurred. Sometimes the thrombi are calcified, and are then known as phleboliths. At the commencement of the affection, before the varices become prominent, fine ramifying vessels are found under the skin, which later on appear between the veins. These ramifying vessels give the skin a brownish appearance.

Varices which extend in the form of ramifying anastomoses and networks over the whole leg are connected both with the skin and subcutaneous tissue, and become very extensive when the valves of the large saphenous vein are destroyed, thus impeding the circulation. The insufficiency of the valves can be shown by raising the limb till the varices have emptied themselves of blood; then compress the saphenous vein at its opening into the femoral vein in the thigh, lower the limb and suddenly remove pressure on the saphenous vein; the varices then become again filled with blood from the femoral vein.

The patients suffer more when standing than when walking. The chief symptoms are tingling and numbness in the limb, cramps in the calves, especially when the deeper veins are affected, swelling of the feet, eczema, ulceration and even elephantiasis. These troubles often cause much suffering.

Varices may be dangerous from rupture and hemorrhage. As a rule the small, thin, ramifying peripheral vessels rupture, sometimes the larger trunks. The blood being under considerable pressure spurts out in a jet. Fatal hemorrhage may take place unless the limb is elevated and the bleeding stopped by pressure. Death may occur in rupture of subcutaneous varices in the leg and in the internal organs (*e.g.* brain and liver). The second danger is thrombo-phlebitis which may lead to embolism, especially when it becomes purulent (Fig. 84).

Differential Diagnosis. Varicose veins are so typical in appearance that they cannot be mistaken for the vascular formations, such as aneurism, circoid aneurism or cavernoma. Primary phlebectasis must not be confounded with the dilatation of superficial veins caused by thrombosis of the deeper veins; *e.g.* after infective diseases.

Treatment. Prophylactic treatment consists in avoiding long standing, in cleanliness and massage. In slight cases the circulation of the limb can be improved by the application of flannel bandages from the toes upwards (*Martin's* rubber bandage is liable to cause eczema). If the varix is caused by pressure of a tumor, this must be removed when possible.

The most radical treatment consists in extirpation of the varices, especially when very tortuous. If the valves of the vein are destroyed (shown by the method mentioned above), it is best to ligature the saphenous vein near its opening into the femoral vein, and to resect a part of it as well. After the operation small varices and eczema quickly disappear, but elastic bandages should be worn for some time. The extirpation of secondary varices due to thrombosis of the deeper veins is useless.

Varicocele should be excised in its whole extent; the testicle can be drawn up by suture.

Submucous varices of the esophagus and varices in the brain and liver are inaccessible to treatment.

Fig. 83 shows somewhat extensive varices in the region of the large saphenous vein in the leg, in a woman of forty, after many pregnancies. The above-mentioned ramifying vessels are seen between the varices, giving the skin a reddish-brown appearance.

In this case the foot was in a position of pronation and abduction (*pes valgus* or flat-foot)

PES VALGUS OR FLAT-FOOT

The treatment of pes valgus depends on its cause. The deformity may be congenital or acquired (traumatic, paralytic, rickety (Fig. 65), or due to long standing).

In all these forms the foot is more or less in a position of pronation and abduction, and eventually there is displacement at the astragalo-scaphoid articulation. Along with changes in the bones and destruction of cartilage in the joints, the ligaments, tendons and muscles are also affected.

Traumatic flat-foot occurs not only after fractures of the leg and ankle, but also as the result of rupture of the ligaments from twisting of the foot, especially when the injury is not treated by fixation. Paralytic flat-foot occurs after acute anterior poliomyelitis, in which the plantar flexors are paralyzed and there is over-action of the extensors.

Rickety flat-foot is due to sinking of the arch of the foot owing to softness of the bones.

The commonest form is static flat-foot, which occurs in persons of weak muscular power, as the result of prolonged standing (waiters, etc.). It generally develops at the age of puberty. The symptoms are fatigue, pains in the ankle and tarsal joints, and on the outer side of the leg. The pains are often cramp-like (tarsalgia).

Differential Diagnosis. Pes valgus must not be confounded with the flat-foot which occurs in certain races (Jews, negroes). The latter is due to imperfect development of the arch of the foot, but there are no

changes in the mid-tarsal joint, and the condition causes little trouble.

Treatment. In congenital flat-foot the position can be corrected by manipulation and massage. In traumatic flat-foot caused by fractures and sprains, the patients should not walk too soon, and then only with a well-made boot provided with a flat-foot sole. In more severe degrees of traumatic flat-foot, the question of cuneiform osteotomy of the scaphoid bone or head of the astragalus, or linear osteotomy of the tibia and fibula may arise. These operations may be considered in cases where manipulation has failed to correct the position. If the tendon Achilles is much shortened it should be tenotomized before manipulation. After manipulation the foot should be put up in plaster of Paris in an over-corrected position.

In paralytic flat-foot tendon-transplantation is useful. The peripheral end of the divided tendon of the paralyzed tibialis anticus muscle can be connected with the tendon of the healthy extensor longus hallucis muscle.

Inflammatory flat-foot, which causes painful contracture, should be treated by rest in bed and hot fomentations. If the pain is very severe cocaine may be injected.

General treatment consists in strengthening the muscles (tibialis anticus and posticus, and calf muscles); active movements and massage. When standing the toes should be turned inwards, and when walking the foot should not be turned outwards. The boots should be well-made with flat-foot pads; the latter are made after an impression of the foot taken on smoked paper, and should extend from heel to toes over the whole sole.

PYOGENIC INFECTIONS

Plate LXVI et seq.

The bacterial invasion of injured or uninjured parts of the body plays a great part in surgery, as there is always the possibility of bacterial infection in every injury and operation.

According to the nature of the infection, definite clinical pictures are produced which are generally represented by various degrees of inflammation and reaction of the body. These processes may be incited not only by bacterial irritation but by mechanical irritation, such as trauma without infection, also by chemical irritation (*e.g.* poisons of all kinds, animal poisons such as snake poison), and by the action of heat and cold (burns and freezing).

In bacterial infection the inflammation is most marked, as it does not remain limited to the place of origin, but extends more or less rapidly in the surrounding parts, and may eventually reach remote parts of the body by way of the blood and lymphatic vessels (general infection). According to the rate of its extension, the inflammation may be acute, chronic or subacute. All three forms may pass into each other.

Bacterial infection causes various clinical phenomena according to the nature, number and virulence of the bacteria, and according to the parts of the organism which are invaded, and the power of resistance of the individual. Old, feeble and diseased bodies (*e.g.* diabetes) are less capable of combating bacterial invasion, while a healthy body shows a strong reaction against it. This reaction

manifests itself by inflammation at the point of infection.

This inflammatory reaction is manifested by the cardinal symptoms—redness, heat, swelling and pain. The redness and heat are due to dilatation of the blood-vessels from irritation of the tissues (active or arterial hyperæmia); the swelling and pain are due to the transmigration of blood elements, especially leucocytes, owing to the slowing of the blood stream. In every severe infection the function of the part concerned is also interfered with.

The exudation varies in degree according to the nature of the infection. It may be serous, fibrinous, sero-fibrinous, or purulent; and when mixed with red blood corpuscles becomes hemorrhagic. Purulent exudation is the most common, and recurs in its simplest form in wounds which do not heal by primary union.

Pyogenic infections are also distinguished according to their situation and extent. They may thus be superficial or deep; circumscribed or diffuse; cutaneous, subcutaneous, muscular, glandular, or osseous, etc.

Besides the local inflammatory reaction of the part of the body attacked, there is a general reaction shown by considerable and prolonged rise of temperature. This must be distinguished from the slighter degree of so-called aseptic fever which occurs during resorption of blood effusions. The temperature chart in pyogenic infections, together with the local reaction and the general symptoms (rigors, pains in the joints, dry tongue, sweating, diarrhea and vomiting) are of the greatest importance in estimating the degree of wound infection.

After the first stage of inflammation, which causes more or less destruction of tissue, comes the stage of regeneration.

Owing to the formation of granulation tissue from the fixed connective tissue cells the inflammatory

area becomes isolated and demarcated, the necrosed tissue becomes separated and is discharged with the pus, and the wound eventually heals by scar tissue which is developed from the vascular granulations. As the stage of reparation proceeds, the clinical symptoms of inflammation subside.

If the infection is very virulent, the body cannot overcome the bacteria and their products of metabolism. From the local infection arises a general infection which the defensive power of the body is generally unable to combat.

The researches of *Ehrlich* and *Morgenroth* have thrown much light on this complicated process. This is not sufficiently explained by the presence of a substance (called alexin) present in the blood-serum, nor by *Metchnikoff's* theory of phagocytosis (destruction of bacteria by the white blood corpuscles), but depends on the combined action of several factors. Also, the still more complicated processes of the formation of antitoxins, and the immunization of the organism, have been made comprehensible by *Ehrlich's* "side-chain" theory.

Again, the knowledge of surgical infections due to bacteria has been extended by numerous observers (*Koch, Fehleisen, Rosenbach* and others). The harmful action of bacteria is due to their multiplication in the organism, and to the formation of products of metabolism, the most dangerous of which are the toxalbumins (or toxins) excreted by living bacteria; while the poisons found within the bacteria, which lead to their destruction, are known as endotoxins and are of less importance.

While the normal skin and mucous membranes only rarely harbor bacteria, every wound forms a favorable soil for their development, and from this they spread by the blood and lymphatic vessels. The organism may be infected by one or several kinds of bacteria (mixed infection).

The most important bacteria from the surgeon's

point of view are those which cause pyogenic infections—the *staphylococcus aureus* and *albus* and the *streptococcus pyogenes*. Most acute inflammatory processes, whether a wound is present or not, are due to these forms of bacteria.

Staphylococcal infections are very common (furuncle, carbuncle, osteomyelitis, etc.), and generally lead to circumscribed purulent inflammations. Streptococcal infections are more diffuse and often cause general infection.

Both these forms of bacteria are especially virulent when they give rise to pyogenic infection of the human body. Other bacteria only cause a slighter degree of inflammation; generally serous or serofibrinous, only occasionally purulent (pneumococcus, typhoid bacillus, bacterium coli commune, gonococcus, bacillus pyocyaneus, tubercle bacillus, diphtheria bacillus).

Differential Diagnosis. Pyogenic infections present such characteristic clinical symptoms that a general diagnosis is not difficult. A stricter diagnosis depends on the history of the case, the local and general condition and bacteriological examination.

Prognosis. With early diagnosis and appropriate treatment the prognosis is favorable as regards life, but doubtful as regards function in certain regions. There is always danger to life in every pyogenic infection, as a circumscribed inflammatory focus may become diffuse and set up general infection. In consideration of this fact, every apparently insignificant pyogenic affection must be treated with the greatest care.

Treatment. In the first place all sources of irritation must be removed (foreign bodies, stone, etc.). Whenever signs of suppuration appear, the affected part must be kept at rest; in the extremities by sus-

pension. When there is inflammatory infiltration of the skin without any formation of pus, it may be smeared with ointment; but the application of an ice-bag is injurious, as it delays the localization of the process in the form of a circumscribed collection of pus, which is the object desired. Hot, moist fomentations are best avoided, as they favor the growth of bacteria. When a circumscribed collection of pus has formed, it must be evacuated by a free incision (pyogenic conditions which require earlier incision will be mentioned later). Small abscesses can be opened under local anæsthesia, but more extensive ones require a general anæsthetic. Local anæsthetics should never be injected into inflammatory tissue, as they are very painful and may also give rise to general infection.

Large incisions, made so as to give the best outlet for the pus, lead to more rapid healing than small incisions. The after-treatment is rendered much simpler by large incisions, while small incisions often require further incision. For the same reason, evacuation of pus by an aspirator is more uncertain and uncleanly.

After-treatment consists in loosely plugging the wound with dry iodoform gauze, and later with sterile gauze, applied daily. Immobilization should be continued till all signs of inflammation have subsided.

In cases where dry tampons cause pain they may be replaced by moist tampons with two per cent. boric acid lotion, one per cent. aluminium acetate, or three per cent. oxygenated water, renewed two or three times a day. Tampons should not be left in too long, as they cause irritation of the tissues. They must, therefore, be managed as carefully as possible, if necessary, under an anæsthetic. The application of alcohol, iodine, carbolic acid, balsam of Peru to infected wounds (cf. treatment of tetanus, Fig. 78), is not to be recommended, as they cause much irrita-

tion in the wound. *Von Bergmann's* method of dry antiseptic dressings is the simplest and most practical method of dealing with pyogenic infections.

Granulation tissue should be treated by ointments of zinc oxide or nitrate of silver, and by baths. Later on, massage, active and passive movements and electricity are indicated, according to the situation and nature of the affection. The general condition also requires treatment in every pyogenic infection, by tonics and nourishing diet. When necessary subcutaneous injections of normal saline solution and nucleinic acid should be given (20 cc. of nucleinic acid in 200 cc. of normal saline solution). Antitoxic or bactericidal serums have so far given no result in pyogenic infections.

The method of passive hyperæmia advocated by *Bier* for the treatment of acute pyogenic infections has, after the experimental and clinical research of *Lexer*, *Wrede*, *Wollf-Eisner* and others, proved itself to be "a double-edged sword." (Discussion at the Surgical Congress, 1906). It cannot be recommended as a practical method, as it necessitates prolonged internment of the patient in hospital. It is true that an increase in the power of defense takes place at the seat of infection after passive venous hyperæmia, as it does after active hyperæmia induced by painting with iodine or hot-air treatment. On the other hand, nutrition is impaired, and the resorption of the bacteria and their poisons delayed by the venous hyperæmia, which may result in further destruction of tissue at the seat of infection. Again, if the infection is a virulent one, especially streptococcal, there may be rapid resorption of bacterial poisons in the organisms after removal of the elastic compression, which may be fatal. In infection by gas-forming bacteria, which may cause gangrene of the tissues by pressure of gases, passive hyperæmia only aggravates this action.

We, therefore, consider treatment by passive hyper-

æmia (which cannot be endured by many patients) as unnecessary in the milder forms of pyogenic infection. The above-mentioned treatment is sufficient in these cases, especially when combined with immobilization. Again, treatment by passive hyperæmia often obscures the indications for incision. Small incisions are often insufficient even in mild cases, and require to be enlarged or repeated, thus complicating and lengthening the treatment. (For the treatment of suppuration in tendon-sheaths, see Fig. 96).

In the more acute pyogenic infections, which present severe clinical symptoms and have a tendency to progress, treatment by passive hyperæmia is unsafe, and has often aggravated the condition; *e.g.*, by thrombo-phlebitis of the small veins, multiple abscesses, and even general infection.

Finally, the treatment of acute pyogenic infections by passive hyperæmia has not a scientific foundation on bacteriological research, nor is it supported by the results of clinical experience.

THROMBOPHLEBITIS ACUTA PURULENTA

(*Acute purulent Thrombo-phlebitis*)

Plate LXVI, Fig. 84.

Acute purulent thrombo-phlebitis may arise from infection of the neighboring parts. In every pyogenic infection purulent thrombi are found in the smaller veins. In the larger veins it arises from periphlebitis, in which there is infection of the wall of the vein. Infection of the walls of veins may also result from internal infection by the blood. Purulent phlebitis always results in the formation of a thrombus which may cause complete occlusion of the vessel. The thrombus generally contains pus (thrombo-phlebitic abscess); it may extend and infect larger areas, or may disintegrate and give rise to general infection by embolism (cf. Fig. 108).

Various pyogenic affections may give rise to thrombo-phlebitis (lymphangitis, furuncle, carbuncle, erysipelas, varicose ulcer of the leg). Otitis media may cause thrombo-phlebitis of the lateral sinus. In the portal vein, infection by the blood may cause pylephlebitis and subsequent multiple abscesses in the liver. Carbuncle of the lips may cause meningitis through thrombo-phlebitis of the facial and ophthalmic veins. When the lesion is superficial, it gives rise to all the symptoms of purulent inflammation—redness, swelling and œdema of the skin and subcutaneous tissue, pain, fever and rigors. The skin is often tense and hard. The infiltration extends along the course of the veins, in the form of hard cords. The presence of pus and the formation of abscess is indicated by yellowish coloring of the skin (Fig. 84), and later by fluctuation.



Fig. 84. Thrombophlebitis purulenta acuta.

Thrombo-phlebitis of the deeper veins gives rise to severe symptoms—pain, high fever, rigors and change in the general condition.

Thrombo-phlebitis of the femoral vein, occurring in women as the result of puerpural parametritis, is known as *phlegmasia alba dolens* (white leg). In this affection the whole leg is affected by painful, hard œdema, preventing any movement. The thrombosis may be so extensive as to cause gangrene of the extremity.

In every case of thrombo-phlebitis the walls of the veins remain thickened causing congestion which, in the lower extremities, leads to deficient nutrition (ulcer, eczema, elephantiasis). Thrombi may become transformed into hard, painful phleboliths, by deposit of calcareous salts.

Differential Diagnosis. Superficial thrombo-phlebitis differs from lymphangitis in the veins being thicker and harder. Deep thrombo-phlebitis is often impossible to distinguish from other pyogenic affections.

The prognosis is always doubtful, owing to the possibility of general pyogenic infection.

Treatment. In the early stages suppuration of the thrombi may be avoided by rest. In the extremities, these should be suspended. The treatment must be conducted according to the general rules for pyogenic affections. Abscesses must be incised; there is no fear of hemorrhage owing to thrombosis of the vessels for some distance from the seat of inflammation. If general infection appears to be imminent the vein should be resected after double ligation of the diseased section. For example, ligation of the internal jugular vein is indicated in otitis media, and in furuncle of the lips (in the latter, also, ligation of the anterior facial vein).

Phlegmasia alba dolens does not suppurate as a

rule and can be treated by rest in bed and the application of mercurial or silver ointments (unguentum cinereum and unguentum Cr  d  ).

Fig. 84 shows acute purulent thrombo-phlebitis in a woman, affecting a varicosity of the saphenous vein, which developed after pregnancy. There is diffuse redness, with yellowish nodules indicating the commencement of abscesses in connection with the infiltrated and thrombosed vein.



Fig. 85. Abscessus subcutaneus.

ABSCESSUS SUBCUTANEUS PARAMAMMILLARIUS

(*Subcutaneous paramammillary abscess*)

Plate LXVII, Fig. 85.

The term abscess is applied to a circumscribed collection of pus which arises from loss of tissue. The terms purulent exudation or empyema are applied to collections of pus which form in pre-existing cavities (maxillary antrum, pleura, abdomen). Abscesses may occur in the skin, subcutaneous tissue, muscles, bones, and also in the internal organs (liver, lungs, brain).

Cold abscesses, which are due to chronic infections, such as tuberculosis (Fig. 125), must be distinguished from acute abscesses, which, in most cases, occur in the subcutaneous tissue as the result of acute pyogenic inflammation, due to staphylococci and streptococci. The formation of an abscess may usually be considered a favorable sign, as it arrests the progress of infection in the organism by damming up the inflammation. After the diffuse inflammation has become circumscribed in the form of abscess, the severe inflammatory symptoms subside. Granulation tissue formed by the fixed connective-tissue cells forms a continuous boundary known as the abscess membrane.

The majority of abscesses arise from diffuse, infiltrating, purulent inflammation of the subcutaneous tissue. Abscesses also occur in the various organs of the body in all other pyogenic affections (erysipelas, lymphangitis, osteomyelitis, lymphadenitis, myositis). The abscesses may spread from the deeper parts to the surface, or inversely.

Blood effusions may suppurate and form abscesses if another part of the body is invaded by bacteria (*e.g.* furuncle). The so-called embolic or metastatic abscesses are formed by way of the blood stream in general infection, and may occur in any part of the body.

The clinical symptoms are those already mentioned. In subcutaneous abscess the skin is at first red, and shows diffuse inflammatory infiltration. There is pain, tension and fever. The red color of the skin becomes gradually darker and more circumscribed. The skin becomes thinner and yellowish and bulging at one spot, through which the abscess bursts. The deeper the abscess, the more diffuse and extensive are the infiltration and inflammatory oedema (*e.g.* in osteomyelitis, Fig. 82).

Erysipelas, lymphangitis, and other pyogenic affections may be present along with abscess formation. The part of the body affected is stiff and painful on movement, and as every abscess may lead to general infection all movements should be avoided.

Differential Diagnosis. Acute abscess is recognized by the presence of all the symptoms of acute inflammation. The cause of the abscess must be found, and the occurrence of metastatic abscesses must be borne in mind.

Treatment. As soon as an acute abscess is diagnosed by the presence of fluctuation, or by an exploring syringe in the case of deep abscess, it must be freely opened. When the suppuration is once circumscribed, early incision prevents further destruction of tissue, leads to quicker healing and leaves less scar. Treatment by hot fomentations or poultices, to cause spontaneous bursting of the abscess, causes more destruction of tissue and delays healing.

After incision the abscess should be plugged with sterile gauze, after which granulation tissue is quickly

formed. Treatment by aspiration is not so good as it does not remove the abscess membrane.

Deep abscesses must be freely opened, plugged and drained. In large abscesses a counter incision should be made at the deepest part of the abscess cavity, and all recesses should be opened up. The affected part should be then immobilized.

Fig. 85 shows a subcutaneous abscess surrounding the nipple in a lying-in woman, arising from a cracked nipple, which gave entrance to bacteria. The skin round the nipple is bluish red and swollen. The presence of fluctuation indicates a collection of fluid in the subcutaneous tissue. The inflammation has already become circumscribed. In spite of the apparently slight extent of the abscess, the patient suffered from severe pain, fever and general *malaise*. The abscess healed quickly, after incision and plugging and suspension of both breasts.

MASTITIS PUERPERALIS PURULENTA

(*Purulent puerperal mastitis*)

Plate LXVIII, Fig. 86.

Bacterial inflammation of the breast (phlegmonous mastitis) ending in suppuration (purulent mastitis), occurs almost exclusively in women during the puerperium, as the result of direct infection of the lactiferous ducts with bacteria (mostly staphylococci), through cracks and fissures of the nipple. The clinical symptoms are those of pyogenic infection, with the formation of a hard, painful infiltration, usually in the lower and outer quadrant of the breast. The skin is tense, oedematous, reddened and often glistening. The redness quickly extends over the whole mamma and beyond it. The patients suffer from a feeling of tension in the breast, and radiating pain in the arm of the affected side. There is also general *malaise*. The affection is often ushered in by rigors and high temperature.

The axillary glands may be enlarged and painful. In severe cases there is diffuse infiltration of the whole mammary gland, which may extend into the lymphatic vessels round the breast. Abscesses form in one or more places; the superficial ones being recognized by fluctuation, the deeper ones by the extensive nature of the lesion. Purulent inflammation of the mamma may occur in general infection; on the other hand, it may also give rise to general infection by thrombo-phlebitis.

Differential Diagnosis. A non-bacterial inflammation of the breast occurs in sucklings soon after birth (mastitis neonatorum). This is a physio-



Fig. 86. Mastitis puerperalis purulenta.

logical swelling of the gland with excretion of a secretion resembling milk. In some cases there is circumscribed abscess formation, which soon heals after incision. The inflammation, however, usually subsides under ointments and moist fomentations. Similar mastitis may occur at the age of puberty, both in boys and girls, which yields to the same treatment and seldom leads to abscess. Pigmentation of the areola remains after these cases of mastitis.

During the period of lactation, accumulation of milk due to stopping its outflow may cause hard inflammatory infiltration of the breast (milk abscess) which disappears after removal of the milk by a breast pump, etc. In these cases both breasts should be supported by a suspensory bandage.

Mastitis may be caused by trauma, by suppuration in a blood effusion caused by injury. In cases of furunculosis and diabetes mastitis may occur, with the formation of hard, deeply situated abscesses resembling malignant tumors.

Tuberculous mastitis is generally due to extension from tuberculous axillary glands and is characterized by its chronic course. Actinomycosis gives rise to hard swellings (cf. Fig. 115). Syphilis may also cause interstitial mastitis, but there is no suppuration. Gonorrheal infection of the lactiferous ducts has also been described, as the result of uncleanness of the mother, or gonorrheal stomatitis in the infant.

Interstitial mastitis and chronic cystic mastitis which form tumor-like nodules in the mamma, cannot be mistaken for phlegmonous mastitis as they cause no acute inflammatory symptoms.

Superficial abscesses in the region of the nipple (Fig. 85) are easily distinguished from purulent mastitis, and are only of limited extent. Retro-mammary abscesses may cause difficulty in the diagnosis when there are also signs of inflammation in the mamma. In these cases the skin is usually intact,

FURUNCULUS—LYMPHANGITIS (*Furuncle—Lymphangitis*)

Plate LXIX, Fig. 87.

FURUNCULOSIS (*Furunculosis*)

Plate LXIX, Fig. 88.

Bacterial invasion of the skin occurs through the ducts of the sebaceous glands. Even slight friction is sufficient to cause staphylococci, which are always present on the skin, to enter the sebaceous glands, where they find more favorable conditions for their growth than on the surface of the skin. In uncleanly persons pustules often occur on the skin, each one pierced by a hair. This purulent inflammation of the sebaceous glands is called *folliculitis*. In the eyelids folliculitis of the eyelashes forms *hordeolum*, or sty. Folliculitis is cured by epilation of the hairs, and may be avoided by cleanliness.

The inflammation may extend beyond the sebaceous gland and cause inflammatory infiltration of the skin. Furuncle (boil) is a circumscribed pyogenic affection of the skin caused generally by staphylococci, sometimes by streptococci and other bacteria. The pathological process consists in hyperæmia and exudation, with redness and hard swelling of the skin, followed by necrosis of the tissue in the center of the infiltration; afterwards regeneration by the formation of granulation tissue. Furuncles occur especially in parts which are exposed to irritation—the nape of the neck, the wrist joint, the buttocks, the thigh and the face. Furuncles often occur secondary to cracked conditions of the skin caused by eczema, excoriations, etc. In diabetics, furuncles are very common owing to the dry condition of the skin and the scratching produced by pruritus, also to the



Fig. 87. Furunculus - Lymphangitis.



Fig. 88. Furunculosis.

body being especially vulnerable to bacterial invasion (Fig. 140). Furuncles may also appear in all cases where the bodily resistance is impaired—in children, old people, and the tuberculous.

The clinical appearance of furuncle is typical. From a small punctiform redness develops a hard, redder, painful nodule in the skin, which extends at its periphery and also deeply towards the fascia. The epidermis is at first intact, but afterwards ruptures at the apex of the projecting furuncle, exposing a yellowish center which becomes more and more demarcated from the hard, red infiltration. In this way a round, crateriform ulcer is produced with a central yellowish core (Fig. 87). Sometimes a hair is situated in the center of the furuncle. Large furuncles are extremely painful, especially on movement, and are often accompanied by fever and general debility. The symptoms subside when the central core becomes loosened by suppuration. The cavity is then quickly filled by granulation tissue, which may form a cicatrix in a few days. The hard infiltration remains for a long time and generally causes unpleasant itching of the skin. The scar, which is always hypertrophic in all inflammatory processes, may also cause trouble.

Complications may increase the severity of furuncle. There is always lymphangitis, especially in the extremities, and often lymphadenitis. Early implication of the lymphatics signifies extensive inflammation and virulent bacteria.

Several furuncles are sometimes found close together, either from simultaneous infection of several sebaceous glands or from secondary infection from the primary furuncle. This often occurs after the application of plaster or other measures with the object of "drawing out" the furuncle.

In individuals with a feeble power of resistance (diabetics, infants and old people), there may be an outbreak of furuncles over the whole body, a condi-

LYMPHANGITIS

In pyogenic affections the lymphatic vessels and glands exercise a beneficial function by harboring and destroying bacteria and their products. If the bacterial invasion is very severe, or the bacteria very virulent, the lymph is coagulated and inflammation takes place in the walls of the lymphatics, first as hyperæmia, later as small-celled infiltration of the walls of the vessels. Virulent bacteria may give rise to lymphangitis and lymphadenitis (Fig. 110) through slight abrasions of the skin, or in connection with pyogenic affections, such as whitlow, furunculosis, etc.

Lymphangitis is most clearly observed in the superficial lymphatics of the extremities, in the form of red, diffuse patches, which soon develop into irregular red cords extending from the periphery to the root of the limb. The number of cords diminishes in the upper part of the limb, and eventually only one large cord remains in the region of the lymphatic glands (inguinal or axillary). These signs are most marked in infection by virulent bacteria.

The lymphatic cords are somewhat raised above the level of the skin and feel hard. They are painful to touch and on movement. There is also itching and a feeling of tension in the whole limb. The regional lymphatic glands are at the same time swollen and painful. In some places abscesses form in the hard cords. There is generally fever and rigors.

Lymphangitis of the deep lymphatics of the extremities can be recognized by the feeling of tension and the general symptoms. Peritonitis may give rise to

pleuritis through the lymphatic vessels of the diaphragm.

The prognosis of lymphangitis is generally favorable, as it disappears after removal of the cause.

Chronic lymphangitis, caused by long-continued irritation of the skin, eczema, ulcers, etc., gives rise to hard, cord-like formations, which persist for a long time. Obliteration of the lymphatics may cause elephantiasis.

Differential Diagnosis. Similar symptoms are caused by acute purulent thrombo-phlebitis (Fig. 84), but the cords are thicker and not so numerous.

Treatment. This consists in treatment of the primary affection which causes the lymphangitis (furuncle, etc.) and in absolute immobilization of the limb, with suspension. The thickened lymphatic cords may be painted with mercury or silver ointments (unguentum cinereum, unguentum Cr  d  ), but these should not be forcibly rubbed in. In chronic lymphangitis, baths and massage are indicated. Abscesses must be incised.

Fig. 87 shows a furuncle with lymphangitis. It was cured in eight days by incision, iodoform gauze, plugging and suspension of the arm.

Fig. 88 shows a case of furunculosis in a young child. Abscess formation is seen in the center of the furuncles. The case was cured by incisions and almond bran baths.

CARBUNCULUS (*Carbuncle*)

Plate LXX, Fig. 89.

Carbuncle, which generally occurs in middle life, differs from furuncle only its greater extent, both superficially and deeply. It consists of an infection of several sebaceous glands, thus forming an agglomeration of furuncles. The skin gives way in several places and there are several yellow cores. Commencing as a small, red nodule, it quickly develops into a hard infiltration, extending to the fascia, and may eventually attain the size of a hand, and cause more or less diffuse inflammatory infiltration of the neighboring parts. Lymphangitis and lymphadenitis are generally present. The affection is accompanied by severe pain, high fever and rigors.

Carbuncle is generally caused by streptococcal infection. Eczema and other affections of the skin which cause furuncle, may also give rise to carbuncle. Moreover, furuncle may develop into carbuncle, especially when the core has been forcibly expressed, or when hot fomentations have been applied. In diabetics carbuncle is still more common than furuncle, and leads to extensive necrosis of the fascia; it often causes death from exhaustion. Carbuncle of the face is dangerous owing to its liability to cause general infection, or meningitis by infection of the facial vein. Carbuncle of the nape of the neck may attain enormous size, and extend from one ear to the other.

Differential Diagnosis. Anthrax (malignant pustule) differs from carbuncle in the presence of small vesicles filled with turbid fluid and early cen-



Fig. 89. Carbunculus.

tral necrosis of the skin, and in the absence of cores. In doubtful cases a bacteriological examination must be made.

Treatment. Under an anæsthetic, a crucial incision is made through the whole extent and depth of the carbuncle, and the central necrosed parts excised. The wound is plugged with iodoform gauze.

In diabetic carbuncle, progressive necrosis of the fascia often necessitates counter-incisions. Iodoform gauze should not be used in these cases, but sterile gauze. Special treatment is required for the diabetes.

In every carbuncle there is severe constitutional disturbance which requires general treatment by nourishing diet, etc.

Fig. 90 shows a carbuncle of the nape of the neck in a patient of forty. The infiltration is very extensive. In the central parts the skin is ruptured in several places, and shows the deeply situated, necrotic cores. Round this is a zone of reddish-blue skin, and beyond this zone an area of hard, red infiltration. There was high fever. The case healed under the above-mentioned treatment.

ERYSIPELAS ERYTHEMATOSUM (*Erysipelas*)

Plate LXXI, Fig. 90.

While in lymphangitis the deeper and larger lymphatics are infected, in erysipelas the smaller lymphatic spaces of the skin and subcutaneous tissue are plugged with streptococci. A similar condition may occur in the superficial layers of the mucous membranes. The causes of this bacterial infection are streptococci (*Fehleisen*), but their identity with the *streptococcus pyogenes* is not yet agreed upon.

The affected skin is red, tense, somewhat glistening and slightly raised above the level of the rest of the skin. The borders are well-defined, distinctly raised and zigzag, so that the extension of erysipelas, especially on the face, has been compared to lambent flames. When the disease spreads over the whole body, it is spoken of as migratory erysipelas.

Erysipelas may occur wherever there is a solution of continuity in the skin—after scratches and excoriations, after all injuries and operation wounds. It may also be combined with various pyogenic affections—whitlow and phlegmon (especially staphylococcal phlegmon). Conditions which give rise to constant irritation of the skin, such as lupus, tuberculous fistula, ulcer of the leg, foreign bodies, etc., may also give rise to erysipelas, which is then often relapsing. Relapsing erysipelas of the face and leg may cause elephantiasis. Lastly, erysipelas may arise in general streptococcal infection, and is then always combined with other pyogenic conditions—abscess, phlegmon, etc.

The common form of erysipelas, which consists in a red elevation of the skin, is called erythematous



Fig. 90. Erysipelas erythematosum.

erysipelas (Fig. 90). In bulbous erysipelas the skin is covered with vesicles (Fig. 91). In hemorrhagic erysipelas there is hemorrhage in the skin (Fig. 91). In the great majority of cases there is resolution, but sometimes erysipelas may cause cutaneous abscesses, and in the form of gangrenous, phlegmonous erysipelas may give rise to ulceration and extensive destruction of the skin.

The clinical symptoms of erysipelas are characteristic. The disease usually commences by a rigor, high temperature (40° – 42° C.) and redness of the skin. There is itching and tension in the skin, and tenderness on pressure. There is considerable constitutional disturbance owing to high fever, headache and vomiting which continue while the disease progresses. The temperature falls suddenly, the redness ceases to extend, and the skin, after slight desquamation resumes its normal condition in about a week from the onset of the disease. In relapsing erysipelas the whole process may take place within one or two days. Erysipelas occurs most frequently on the face, after this on the extremities and genital organs. In places where the skin is loosely attached (eyelids, scrotum), there may be considerable swelling and œdema.

Erysipelas of the mucous membranes is generally difficult to recognize, except when it is an extension from erysipelas of the skin. The mucous membrane is swollen, œdematous, sodden and of a deep-red color. Constitutional disturbance is generally severe. Erysipelas of the buccal mucous membrane may occur after tooth extraction with dirty instruments. It may cause death by meningitis or œdema of the glottis. The average mortality of erysipelas is ten per cent.

Differential Diagnosis. Erythematous erysipelas is so characteristic that it can hardly be mistaken for other affections. The advancing, irregular, raised

edge distinguishes it from other inflammatory conditions.

Treatment. The affected parts must be covered with antiseptic ointments to prevent infection and auto-infection. If pain is very severe scarifications are useful. In erysipelas of the extremities the healthy skin, at the upper limit of the lesion, may be painted with a single application of pure carbolic acid, which destroys the superficial layers of the skin. However, in spite of this procedure, the erysipelas often extends further up the limb. The induction of passive hyperæmia, by surrounding the limb with adhesive plaster, has also been recommended. Among other methods, painting with iodine may be mentioned. The patient should always be kept in bed.

Serum therapy has so far proved useless, and is likely to remain so, since repeated attacks of the disease do not confer immunity.

The formerly extolled curative action of erysipelas on tumors has proved illusory. If erysipelas extends over a malignant tumor (carcinoma or sarcoma), the tumor may diminish in size owing to destruction of its cells, but it soon begins to grow again. The same thing occurs after injection of the fluid, and this explains the temporary action of the so-called cancer serum.

On account of the infectious nature of the disease, the patient should be isolated, and the room disinfected with formalin vapor. The same disinfection must be carried out in operation theaters when an epidemic of erysipelas occurs. However, it is more often the hands of the surgeon which convey infection; hence great care must be taken in avoiding contact with the patient as much as possible, and in disinfecting the hands.

Fig. 90 shows a typical case of erythematous erysipelas of the face, which originated from a fissure on

the nose. In a few days there occurred high fever and rigors, followed by erysipelas, first on one side of the face, then on the other. The skin was tense, purple and somewhat raised. There was considerable pain and itching. The eyelids were so œdematous that the patient could hardly open them. The lips were also much swollen, and there was commencing erysipelas of the buccal cavity. The sharp zigzag borders are seen towards the scalp and the neck.

ERYSIPELAS BULLOSUM HÆMORRHAGICUM

(*Hemorrhagic Bullous Erysipelas*)

Plate LXXII, Fig. 91.

This case is interesting on account of the origin of the infection from a horse bite in the arm. Round the three wounds (which were only superficial abrasions) the skin is dark red and there are annular extravasations of blood. There are also several vesicles filled with turbid fluid. There is extensive diffuse reddening, especially on the forearm, and a brownish coloration due to numerous extravasations of blood from the smaller blood-vessels situated round the lymphatic vessels. In the upper arm there is macular and cord-like reddening due to lymphangitis. The axillary glands are much swollen and painful.

Wounds caused by bites from animals or men tend to become severely infected. In this case, the swelling of the forearm was so extensive that a deep phlegmon was suspected. The symptoms quickly subsided after suspension of the arm. In the place where the erysipelas was hemorrhagic and bullous, there occurred a superficial phlegmonous inflammation, which led to gangrene of the skin.

Differential Diagnosis. This has to be made from several other affections. Anthrax also commences with redness of the skin and the formation of vesicles (Fig. 112), fever and rigors, and may, in its early stage, be confounded with this form of erysipelas. But the redness is not so extensive in anthrax, nor so rapidly developed. Anthrax always causes early gangrene of the skin. In doubtful cases

anthrax bacilli must be looked for in the contents of the vesicles.

In this case, which arose from a horse bite, there was a suspicion of glanders. But, in the latter the redness is punctiform or macular; the vesicles are larger and purulent, and soon rupture, giving rise to gangrenous ulcers.

Subcutaneous phlegmons, which arise from very virulent streptococci, may cause an erysipelatous redness of the skin, but this only occurs in the region of the phlegmon, and does not extend so rapidly as erysipelas. Vesicles may also form on the skin in virulent streptococcal infection.

Phlegmons due to gas-forming bacteria (*e.g.* malignant œdema, Fig. 109) cause rapid redness and swelling of a whole limb. Increase of pressure in the tissues from the formation of gas also gives rise to the formation of vesicles, but these are very large and often raise the epidermis over the whole part affected (Fig. 109). In these severe forms of phlegmon there are signs of general infection from the beginning—rigors, delirium, diarrhea, dry tongue, and bacteria in the blood.

In all the above-mentioned cases the clinical pictures may be very similar, and the diagnosis should always be established by bacteriological examination. Correct diagnosis is all the more important to establish, as the treatment differs in the different affections. In erysipelas, anthrax and glanders conservative treatment is indicated, while streptococcal phlegmon requires early incision to prevent general infection and in gas-phlegmon very extensive incisions, or even early amputation of the limb, may be necessary to save the patient's life.

In Fig. 91 streptococci were found in the vesicles, and from this, together with the clinical symptoms the diagnosis was made of hemorrhagic bullous erysipelas; but the possibility of a deep phlegmon

due to the bite still remained. However, the mildness of the constitutional disturbance, and the rapid disappearance of the swelling showed it to be a case of erysipelas only. Recovery took place in the course of three weeks, with cicatrization of the gangrenous part.



Fig. 92. Erysipeloid.

ERYSIPELOID
Plate LXXIII, Fig. 92.

An affection very similar to erysipelas, called chronic erysipelas by *Rosenbach*, is now known by the term erysipeloid. This is also a bacterial infection of the skin (according to *Tavel*, also of tendon-sheaths and joint capsules) but of a very harmless nature. The specific cause of erysipeloid is unknown; in some cases the staphylococcus albus has been found.

The affection begins with redness and swelling of the fingers. Like erysipelas, the redness has sharp, irregular borders. The redness spreads slowly but continuously over the whole finger, and may extend to the next finger and as far as the wrist. At this point the inflammation stops. There are no constitutional symptoms; no fever nor rigors. The patients only complain of itching and a feeling of tension in the skin. In some cases there is lymphangitis, generally on the extensor surface, as far as the axilla. In rare cases lymphadenitis with high temperature has been observed.

Erysipeloid generally occurs after injuries to the fingers, especially by fish and game. It is, therefore, more common in venders of fish and game, cooks, butchers, curriers, etc. Sometimes the injured spot is invisible, as the redness and swelling generally appear a few days after the injury. In other cases foreign bodies are found in the skin. The affection has been observed in doctors after operating upon infected persons. The symptoms generally subside in a week, but relapses are common. The disease is more common in the autumn.

Differential Diagnosis. Erysipeloid differs from erysipelas in its chronic course, absence of fever, paler color, and demarcation at the wrist.

Treatment. Ointments, rest and support on splints. Movements must be restricted after removal of splints, to avoid relapses. Foreign bodies must be removed. Suppuration has never been observed. Baths and iodide ointment may be used if swelling persists.

Fig. 92 shows erysipeloid in a cook, which appeared soon after handling game. A few days after a slight wound, redness and swelling developed at the tip of the right forefinger, and gradually extended over the whole finger. At the base of the finger the edge of the redness is irregular and zigzag.

Panaritium or Panaris

(*Whitlow*)

PANARITIUM SUBEPIDERMIOIDALE (*Sub-epidermic whitlow*)

Plate LXXIV, Fig. 93.

PANARITIUM SUBCUTANEUM (*Subcutaneous whitlow*)

Plate LXXV, Fig. 94.

PANARITIUM OSSALE ET ARTICULARE

(*Osseous and articular whitlow*)

Plate LXXVI, Fig. 95.

PANARITIUM TENDINOSUM (*Tendon-sheath whitlow*)

Plate LXXVII, Fig. 96.

PANARITIUM INTERDIGITALIS (*Interdigital whitlow*)

Plate LXXVII, Fig. 97.

Subcutaneous suppuration in the fingers and toes is called *whitlow*. Although various forms of whitlow are distinguished, this usually begins as an infection of the subcutaneous tissue (primary subcutaneous whitlow), from which may arise tendinous, periosteal, osteal or articular whitlow, according to the extent of the inflammatory process.

Subcutaneous whitlows occur most often in the fingers, especially among the working classes who are subject to cracks and fissures of the skin. They often occur after punctured wounds, through which staphylococci, or more rarely streptococci, gain entrance to the subcutaneous tissue.

The anatomical formation of the subcutaneous tissue is peculiar, vertical connective-tissue septa separating the fatty connective tissue into a number of distinct compartments. If bacteria gain an entry into such enclosed chambers the inflammation they cause is at first circumscribed.

As in all infections, there is hyperæmia, exudation and necrosis of tissue; the latter occurs rapidly, owing

to the impairment of nutrition from pressure in the inflamed area. In this way a necrotic core is formed, as in furuncle. The increase of tension in the tissues causes severe pain, and the finger becomes red and swollen. In horny-handed workmen the seat of infection is at first difficult to see, and is only made evident by the great pain on pressure. Later on, when the suppuration has extended further, the pain is not so circumscribed. In a few cases only, the skin gives way and a yellow core becomes loosened and cast off, after which healing takes place by granulation tissue. The hard skin on the palmar surface of the fingers prevents escape of pus, so that the latter takes paths of less resistance. The vertical connective tissue septa, mentioned above, direct the pus towards the peritendinous tissue, where it may spread along the whole length of the tendon. The pus may also reach the loose connective tissue on the dorsal surface, and give rise to redness, swelling and œdema, while inflammatory signs may be absent at the seat of infection on the flexor surface. If the tendon sheath is bathed in pus for some time it becomes perforated, and the pus extends within the tendon sheath (tendinous whitlow, Fig. 96). In the same way the periosteum, bony cortex, medullary cavity and joint may become infected from a subcutaneous whitlow (Fig. 95).

A further danger of whitlow is spreading of pus to the hand and forearm along the tendon sheaths. General infection may also occur.

The clinical symptoms vary according to the duration and extent of infection, and the virulence of the bacteria. In sub-epidermic whitlow (Fig. 93), a purulent vesicle develops, generally on the dorsal surface, with slight redness of the surrounding skin. The raised epidermis sometimes shows several yellow spots, where the pus breaks through. Pain and functional disturbance are slight, the inflammation remaining local. There is seldom lymphangitis, no

tendency to spread, and little or no constitutional disturbance.

In subcutaneous whitlow it is quite otherwise (Fig. 94). The whole finger is red, swollen, flexed and extremely painful, especially at one spot. Redness, swelling and oedema are often more marked on the dorsal surface, together with lymphangitis of the hand and forearm. There is moderate fever (39° C.) and some constitutional disturbance.

The symptoms are most severe in tendinous whitlow (Fig. 96). There is more swelling of the finger, and the latter is more flexed. There is pain on pressure along the whole tendon sheath, and usually over the whole palm. Movement of the tendon causes great pain, and extension is almost impossible. Lymphangitis and erysipelatous reddening often extend far beyond the seat of infection. There are rigors and rise of temperature (40° C.), sleeplessness, and considerable *malaise*.

If the tendon sheath of the thumb or little finger is infected, the pus may extend along the course of these sheaths as far as the wrist; whereas, suppuration in the tendon sheaths of the second, third and fourth fingers does not extend beyond the metacarpophalangeal joints, where these tendon-sheaths end.

In the wrist the tendon-sheaths become widened and lie so close together that suppuration may extend from one to the other. In this way, infection of the tendon-sheaths of the thumb may result from a lesion of the tendon of the little finger; and inversely, infection of the little finger from the thumb. This has been called V-shaped whitlow. It is obvious that infection of both tendon-sheaths causes severe symptoms—high fever and much constitutional disturbance. The thumb and little finger are flexed, swollen and very painful on pressure. The pus often breaks through the tendon-sheaths and extends between the muscles of the forearm up to the elbow joint, in the form of deep, progressive suppuration.

In other cases the wrist-joint is infected. Such cases may give rise to general infection.

The V-shaped whitlow is recognized by its severe clinical symptoms and typical appearance. In the early stages there is often pain, redness and swelling in the palm, or on the flexor surface of the wrist. When suppuration has existed some time and become extensive it seeks a way to the surface. In this way fistulas are formed in the course of the tendon-sheaths, discharging much pus, and often exposing the greenish-yellow remains of the necrosed tendon (Fig. 96). The orifices of these fistulas are surrounded by flabby, unhealthy granulations which, as mentioned before (Fig. 56), indicate necrosis in the deeper parts.

In periosteal and osteal whitlows, which generally occur at the ends of the fingers, the periosteum and bone are surrounded by pus and destroyed. In the terminal phalanx total necrosis may occur. A fistula forms and discharges the fetid, slimy pus, which is characteristic of necrosed bone. Eventually dead bone is discharged. (Fig. 95). Parts of the skin may become necrosed, so that, eventually, the whole finger-joint may be lost. Commencing with sharp pain, the acute stage gradually becomes more chronic, and in this stage infection of the bones may be overlooked.

In the first and second phalanges there is often infection of the joints, either secondary to infection of the periosteum, or directly from the surface. Articular whitlow generally manifests itself by rigors. The joint is fixed in a position of flexion and is very painful on movement. The capsule and ligaments are soon destroyed, and destruction of the cartilage causes grating on movement. Articular whitlow may give rise to general infection.

It is not always easy to diagnose the stage of the whitlow. Patients of the working class generally come so late for treatment that there is often infection

of the tendon-sheath, periosteum and joint. In other cases the pain is so severe as to suggest tendinous whitlow, while it is only subcutaneous. A correct diagnosis can often only be made after incision.

Differential Diagnosis. Tuberculous and syphilitic inflammations are more chronic and cause less pain and fever. They do not heal after incision, but require specific treatment.

Treatment. All whitlows require early incision. In sub-epidermic whitlow the purulent bulla must be opened and dressed with antiseptic dressings, and the arm suspended in a sling. Sub-epidermic whitlow may cause infection of the deeper tissues, and there is also the danger of erysipelas. Hence, plenty of dressing should be used.

Subcutaneous whitlows should be incised as soon as possible, under an anæsthetic. *Schleich's* infiltration anæsthesia is dangerous and painful in infected areas. However, endoneural injection of one per cent. cocaine may be made in the first phalanx, according to the method of *Oberst-Corning*, if there is no sign of inflammation at this place. But general anæsthesia should be employed in all cases where the extent of the suppuration is not clear. Incision should be made into the subcutaneous tissue on the palmar surface away from the middle line, and between the joints. The wound is then held open by retractors and examination made for pus in the tendon-sheath or under the periosteum. This examination can only be made by a free incision, after the hand is made bloodless by the elastic tourniquet.

This is the safest method of dealing with whitlows; for although some cases may be cured by evacuation of the pus through a small incision, suppuration in the tendon-sheath may be overlooked, and this may lead to spread of suppuration, destruction of tendon, etc., and even death from general infection.

No doubt, infected wounds of the finger often subside with rest in a sling; but sometimes the periosteum is infected, and this conservative treatment then results in necrosis of the phalanx. Therefore, we must urge the treatment of all such infected wounds by early incision, especially in doctors who are liable to virulent infections.

Tendon-sheath whitlows require very careful treatment, in order to preserve the tendon and the function of the finger. Some cases come too late for treatment for the tendon to be preserved. Many cases of tendon-sheath whitlow extend so rapidly, and so often lead to general infection, that they require free incision of the whole area of suppuration. In some cases, no doubt, this may cause injury to or loss of the tendon; but it is far worse to be responsible for a general infection which might have been avoided by more extensive incision. Therefore, in extensive tendon-sheath whitlows, especially in V-shaped whitlows, free incisions are necessary, but these should always be made laterally. In V-shaped whitlows care must be taken to preserve the palmar carpal ligament; this must only be divided when there is threatening infection of the wrist joint, or extension of suppuration up the forearm.

Better functional results are obtained by several smaller incisions instead of one continuous incision. Too much plugging of the wound is to be avoided, as it interferes with the nutrition of the tendon. After-treatment consists in early passive movements.

In osteal whitlow necrosed bone must be removed if present. In the terminal phalanx it is often sufficient to remove the peripheral end. If the joint is much destroyed resection of the bone, or even amputation may be required.

Progressive suppurations, due to infection by virulent bacteria or to extensive injuries, must be freely laid open, sometimes as far as the bone. If general infection supervenes the question of amputation arises.

Although early incision removes the danger of the pyogenic condition spreading by subcutaneous suppuration, this danger may recur if the after-treatment is neglected.

The incisions should be lightly plugged with iodoform gauze, which best absorbs the discharge. After the first dressing this should be replaced by small pieces of sterilized gauze, sufficient to keep the edges of the wound open and allow the pus to escape. The hand and forearm should be immobilized on a splint. Under this treatment even deep cavities begin to granulate in a few days, when the plugging should be left off and replaced by baths and ointment.

To decide the time when plugging may be left off is a matter of experience. If it is kept on too long the nutrition of the tendon (in the case of tendinous whitlow) is impaired. If it is left off too soon, suppuration may extend into the deeper parts; this is manifested by further redness, swelling and pain, and by a fetid, slimy discharge from the wound and the formation of flabby, unhealthy granulations. Increase of pain is often a sufficient sign of fresh infection. If the extension of infection is not immediately noticed it may cause severe complications and general infection, even during the period of after-treatment. This reinfection may be avoided by several measures. First of all, patients with severe forms of whitlow should be treated in hospital, where they can be kept under observation and treated under more favorable conditions.

The temperature, in severe cases of whitlow, should be taken every four hours. The dressings should be changed every day, if necessary under an anæsthetic, so that the local condition can be examined. The gauze tampons should be carefully removed during irrigation with peroxide lotion. The wound should then be irrigated with normal saline solution under very slight pressure, and the dressing renewed. It is often necessary to hold the edges of the wound apart

by retractors, so as to obtain a better view of the condition of the wound, and drain all suspicious pockets. Drains should be only retained after the first change of dressing in extensive whitlows. When the dressings are changed examination must be made for inflammation and suppuration in parts remote from the wound—in the palm in tendinous whitlow, and in the wrist and elbow joints in V-shaped whitlow. Even in the slighter forms of whitlow the dressings should be changed every day, especially in out-patients (polyclinic). This avoids stiffening of the fingers by prolonged immobilization, also the troublesome condition called “glossy skin.”

After-treatment is begun when the suppuration has ceased and the temperature has become normal. This consists in performing passive movements of the fingers each time the dressings are changed. In out-patient practice (polyclinic), large immobilizing dressings should be applied after these passive movements have been performed. This is especially necessary in alcoholic patients, in whom the inflammation is much aggravated. Moreover, small dressings may be removed by the patient himself. The application of large immobilizing dressings has a favorable influence on the inflammation, and renders the after-treatment easier and shorter, while the disadvantage of immobilization is removed by daily passive movements when the dressings are changed.

In the treatment of whitlows it is best to pursue a middle course. On the one hand, too large incisions, too much plugging and too long immobilization cause impairment of function; on the other hand, small incisions, too little plugging and too free movement may lead to general infection. Radical treatment is best for the beginner, although more conservative methods may be adopted with further experience.

After-treatment must be commenced directly suppuration has ceased (massage, baths and passive movement).

Cicatricial contractions sometimes yield to gradual extension; but some cases may require excision of the scar, followed by a plastic operation.

As already mentioned *Bier's* passive hyperæmia treatment is contra-indicated in acute pyogenic affections; in mild cases it is unnecessary, and in severe cases it is dangerous.

Other methods, such as injection of carbolic acid lotion, staphylococcal serum, carbolic and alcoholic fomentations generally do harm.

PANARITIUM SUBEPIDERMIOIDALE (*Subepidermis Whitlow*)
Plate LXXIV, Fig. 93.

In this case a circumscribed redness developed on the dorsal surface of the left forefinger, without any obvious injury. This was followed by the formation of a purulent blister. The epidermis is raised and shows several yellow points due to the presence of pus. The movement of the finger was not impaired. The blister was opened, the pus evacuated and the thin epidermis removed. The wound was dressed with sterilized gauze, and the finger put up on a splint extending above the wrist.



Fig. 93. Panaritium subepidermoidale.

PANARITIUM SUBCUTANEUM (*Subcutaneous Whitlow*)
Plate LXXV, Fig. 94.

This figure shows a subcutaneous whitlow, which is the most common form of pyogenic infection of the fingers; according to *von Bergmann*, it is the first stage in all the other forms of whitlow.

A few days after a slight abrasion of the skin, redness and swelling developed on the dorsal surface of the thumb (the volar surface is most commonly affected). This extended to the volar side, where the color was paler and more bluish. There was also inflammatory reddening on the back of the hand. As there was only slight pain the patient continued to use the arm. After this fever and rigors occurred, with acute lymphangitis extending over the back of the hand and forearm, and lymphadenitis of the axillary glands, so that the patient could no longer use the finger. At the seat of infection the skin gradually became thin and yellow, showing that the pus was about to discharge through the skin. (In subcutaneous whitlow of the volar side this is prevented by the thickness of the skin). The appearance of the lesion at this time resembled a furuncle. Above this there were several purulent vesicles surrounding a circumscribed gangrene of the epidermis caused by oedema. Fluctuation is seldom present in whitlow.

An incision about half an inch long was made on the volar side away from the tendon. The wound was plugged with iodoform gauze. The lymphangitis was treated with ointment, and the whole arm put on a splint. Function of the finger was restored in ten days.

PANARITIUM OSSALE ET ARTICULARE

(*Osteal and Articular Whitlow*)

Plate LXXVI, Fig. 95.

In this case a punctured wound of the tip of the finger was followed by pain, redness, swelling and some fever. It was treated with poultices. The skin gave way at one place, forming a fistula which discharged fetid pus. Part of the necrosed phalanx protruded. The skin above the fistula became gangrenous, and unhealthy granulations formed round the fistula. Owing to absence of operative treatment, the suppuration extended to the joint and destroyed ligaments, capsule and cartilage, so that the function of the joint was destroyed. After further treatment with fomentations, the whole finger became swollen and the skin assumed a pale, glistening appearance (glossy skin), indicating necrosis of the whole basal phalanx.

Under an anæsthetic an incision was made, and the first and second phalanges were found to be so much destroyed that they were removed.

As already mentioned, punctured wounds of the terminal phalanx, beyond the insertion of the tendon, often lead to infection of the periosteum. Commencing in acute inflammation with pain and swelling, they often assume a more chronic condition. If an incision is not made in the acute stage there may be extensive destruction, even of the whole finger; especially after treatment with poultices. In the above case an early incision would have saved the finger and restored normal function.

"Glossy skin" (*Paget*) is a condition which affects chiefly the phalanges of the fingers, after badly cov-



Fig. 95. Panaritium ossale et articulare.

ered amputation stumps, or after too-long immobilization. This condition may extend over the whole finger. The skin is at first thickened, bluish red, and cold to the touch; later on it becomes pale yellow and has an appearance like parchment. The circulation is bad and there are often neuralgic pains and a feeling of coldness. It may finally lead to traumatic neurasthenia. This condition can be prevented by avoiding too long immobilization and by providing the amputation stumps with sufficient well-nourished flaps.

PANARITIUM TENDINOSUM (*Tendinous Whitlow*)
Plate LXXVII, Fig. 96.

This is a case of subcutaneous whitlow, following a punctured wound, which rapidly spread to the tendon-sheath of the thumb. A severe form of infection was indicated by the acute redness and swelling, severe pain, high temperature and constitutional disturbance. As no incision was made, the terminal phalanx continued to swell and finally gave way, forming a fistula discharging pus and parts of necrosed tendon. Apart from this, the diagnosis of tendinous whitlow could be made from the severity of the symptoms; from the complete loss of movement in the thumb, the great pain on pressure over the course of the tendon, the swelling and redness of the ball of the thumb, and the discharge of pus from the fistula on pressure over this part. The tendon sheath of the little finger was unaffected, and there was no sign of abscess above the wrist.

Under an anæsthetic an incision was made along the whole of the terminal phalanx and pus evacuated from the tendon-sheath. A second incision was made in the palm, a little below the wrist, and the tendon-sheath opened again at this point. By this means the suppuration ceased and infection of the tendon-sheath of the little finger was avoided. As the tendon of the thumb was already partly destroyed, the end joint remained functionless. In spite of a certain degree of contracture, the patient could use the thumb, by movement at the metacarpophalangeal joint.

PHLEGMONE INTERDIGITALIS (*Interdigital Whitlow*)
Plate LXXVII, Fig. 97.

This term is applied to subcutaneous suppuration between the metacarpal bones. In Fig. 97 this occurred between the metacarpal bones of the thumb and index finger. Redness and œdema appeared on the dorsal surface and movement of the fingers was painful. In these cases there is usually some fever, but no lymphangitis or constitutional disturbance. As the amount of pus is usually considerable, there is fluctuation. The pus was evacuated by a dorsal incision (incision on the palmar side is to be avoided); the wound was plugged for a short time and the arm suspended in a sling. Complete function was restored.

Early incision prevents spreading of suppuration to the palm. Interdigital whitlow in the palm is distinguished from tendon-sheath whitlow by there being less pain on movement of the fingers, and less tenderness on pressure over the tendons.

PARONYCHIA (*Peri-ungual Whitlow*)
Plate LXXVIII, Fig. 98.

Inflammation of the tissues under the nail is called *sub-ungual whitlow*. Owing to pressure of the nail, the virulence of the infecting bacteria is increased, so that the inflammation extends rapidly and soon leads to necrosis of the tissues. Sub-ungual whitlow causes severe pain and lymphangitis. It is often overlooked, as the changes under the nail are not at first visible, and the first sign is usually a yellow coloring seen under the nail. The diagnosis is suggested by the severe pain on pressure on the nail. As the pus cannot break through the nail, it extends deeply and may cause necrosis of the terminal phalanx by infection of the periosteum. Clavi and exostoses may also develop under the nail and cause inflammation with severe pain. Under local anæsthesia the nail may be pared down with a knife, so that the inflammatory area can be incised. If suppuration is extensive the nail must be removed.

When the inflammation is not under the nail but around the nail bed, the condition is called *peri-ungual whitlow* or *paronychia*. This may be caused by punctured wounds, tearing of the nail, foreign bodies, or by manicure with dirty instruments. The bed of the nail is red, infiltrated and painful on pressure. There is often suppuration round the nail, which is raised from its bed and may become quite loose. In severe cases there is much pain, fever and lymphangitis.

Differential Diagnosis. Syphilitic chancre of the finger often resembles paronychia. It begins with



Fig. 98. Paronychia.



Fig. 99. Unguis incarnatus.

redness and hard infiltration which develops into an unhealthy ulcer with flabby granulations. This is followed by painful infiltration of the lymphatic vessels and glands. This form of chancre is very chronic and painful (thus differing from most other chancres). Syphilitic chancre should be borne in mind in every case of chronic paronychia which is refractory to treatment. It is especially common in medical men and midwives.

Tuberculous infection of the nail bed may also occur among doctors and nurses. This begins in a dark-red infiltration of the skin. Nodules then develop and break down into an ulcer with flat, irregular borders. The tuberculous granulations are grayish red and bleed easily. This affection is very chronic. The nail may be lost and replaced by thickened tissue in both tuberculous and syphilitic paronychia. In some cases the whole finger may be destroyed. The diagnosis of tuberculous paronychia can sometimes only be settled by microscopic examination, or by inoculation of the guinea pig. The diagnosis of syphilitic chancre is confirmed by finding the *spirochaeta pallida* in scrapings.

Treatment. In peri-ungual whitlow or paronychia an early incision should be made, before the pus has loosened the nail. It is best to make a horseshoe incision through the soft parts some distance from the nail, to avoid interfering with its nutrition. The hand should be immobilized for a few days. If the nail is extensively separated it must be removed.

Tuberculous paronychia requires treatment by the sharp spoon or *Paquelin's* cautery. Syphilitic chancre must be treated by mercury.

Fig. 98 shows acute inflammatory infiltration round the nail. The skin is bluish red and tender to the touch. Under local anaesthesia a horseshoe incision was made through the infiltrated tissue. Healing took place with preservation of the nail.

UNGUIS INCARNATUS (*Ingrowing toenail*)
Plate LXXVIII, Fig. 99.

Ingrowing toenail affects almost exclusively the nail of the great toe; generally the outer side, less often the inner side, occasionally both sides. It gives rise to severe inflammation of the soft parts next the border of the nail; first redness and swelling, afterwards ulceration and granulation tissue. The inflammation is usually limited to a small area, but may sometimes spread over the whole nail-bed. The affection causes considerable pain and often prevents the patient from walking. There may be lymphangitis. If both sides of the nail are affected the symptoms are naturally more severe. Ingrowing toenail often occurs in connection with hallux valgus (Fig. 64); it may also be caused by anomalies of the nails or toes, by wearing too short boots, or by cutting the nails too much at the sides.

Differential Diagnosis. Subungual clavus or exostosis may cause inflammation round the nail, but in these cases the nail is always raised in front and is very tender to pressure. Syphilitic chancre has also been known to occur on the great toe, after sucking the toe (*Bockenheimer*).

Treatment. Ingrowing toenail may be avoided by prophylactic treatment. The toenails should be cut straight and not too short, so that the free border extends beyond the soft parts, especially at the sides. Attention should be paid to cleanliness and to the wearing of properly made boots. In slight cases the edge of the nail may be raised from the inflamed soft

parts by an iodoform tampon, or partial excision of the nail may be performed. In severe cases these methods are useless. Excision of the nail, which was formerly practiced, is useless, as the condition recurs after. The most rational method consists in excision of the whole lateral border of the nail together with the inflamed soft parts, down to the bone; taking care to include the posterior part of the matrix, so that recurrence cannot take place. The wound is dressed with iodoform powder and sterilized gauze and immobilized for a week, after which the wound is usually healed. In ingrowing toenail affecting both sides the same operation is performed on each side, leaving the center part of the nail in place.

Fig. 99 shows an ingrowing toenail on the outer side of the right great toe. The thickened soft parts have grown over the border of the nail. There is a purulent discharge from unhealthy granulations. The nail is so imbedded in the swollen soft parts that it is only partly visible. The above operation was performed with good result.

CLAVUS INFLAMMATORIUS (*Inflammatory Clavus*) (Corn)
Plate LXXIX, Fig. 100.

The figure shows an inflammatory condition affecting the whole of the second toe and extending to the dorsum of the foot. The skin on the dorsal surface of the toe was at first raised by purulent vesicles. After these had broken, the necrosed epidermis came away, exposing a considerable extent of the corium. The redness and swelling are most marked over the first interphalangeal joint, which was very painful on movement. On the dorsal side of the joint fluctuation was present. The remains of a clavus (corn) are seen on the great toe, in the form of a yellowish-white projection, together with a fistula leading to the deeper parts. The clavus on the second toe was due to its being exposed to pressure from its crooked position.

Clavi, or corns, are circumscribed growths which arise from the horny layer of the epidermis. They generally occur on the great and little toes; sometimes between the toes, especially when these are crooked owing to bad boots. They also occur in connection with hallux valgus, hammer-toe, club-foot, etc. The more they project above the level of the skin the more painful they are to pressure. They differ from the diffuse, horny thickenings which occur on the hands, and consist in a circumscribed horny formation which develops from a soft conical core situated in the depth of the cutis. When the horny layer is removed the soft yellowish-white core is seen in the center. Lacerations caused by unskillful cutting of corns may easily give rise to subcutaneous abscess. Underneath large clavi there is usually

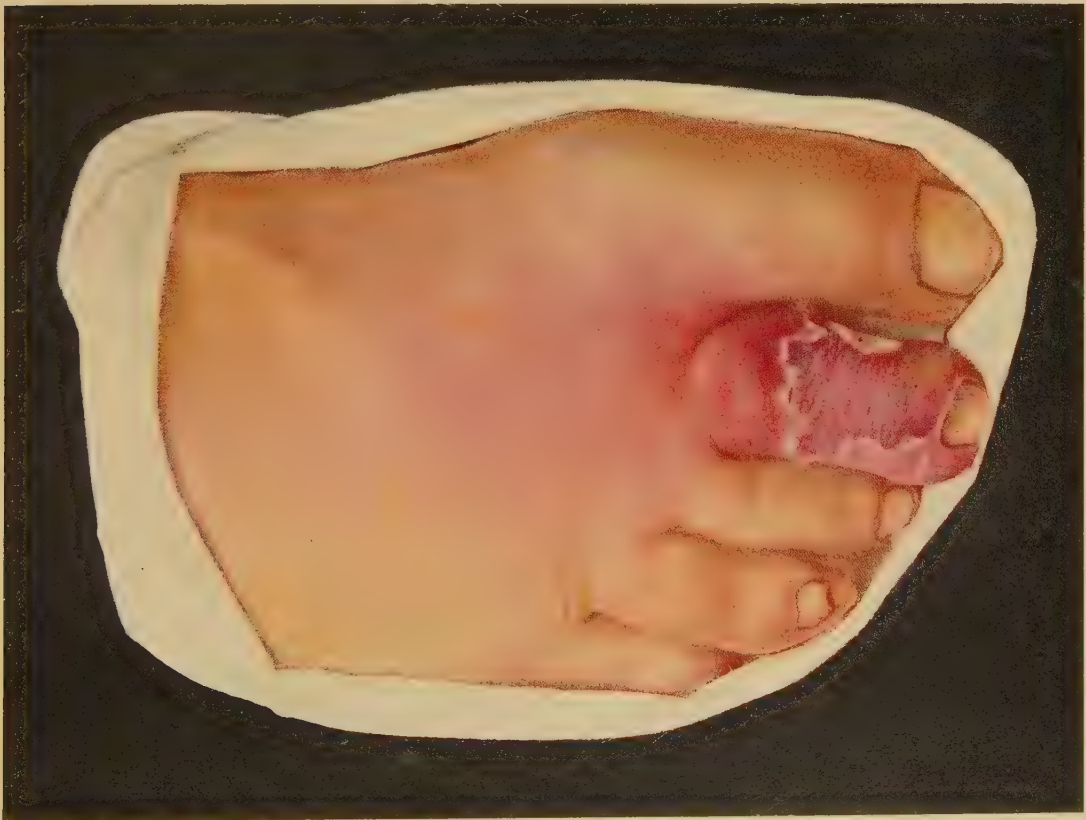


Fig. 100. Clavus inflammatorius -- Arthritis purulenta.



Fig. 101. Phlegmone progrediens putrida.

developed a bursa, which is liable to become inflamed from external pressure. The inflammatory exudation from the bursa generally discharges by a fistula near the clavus (Fig. 100). Septic infection of the bursa may be caused through the fistula, and this may extend to the neighboring tendon-sheath or joint. Joint infection is especially frequent when the bursa communicates with the joint; and is manifested by severe local inflammation, fever, rigors and constitutional disturbance. The purulent arthritis may even give rise to general infection.

Treatment. Prophylactic treatment of clavus consists in cleanliness and the wearing of proper boots. If a clavus forms it should be removed with a sterilized knife. It is not sufficient to remove the horny layer; the deeply situated core must also be removed, otherwise recurrence takes place. Other methods, such as the application of salicylic colloidion, only loosen the horny layer and do not prevent recurrence.

If a bursa forms under the clavus it must either be incised and plugged, or excised. If suppuration extends to the joint this must be opened; in some cases resection or disarticulation may be necessary.

In Fig. 100 there was inflammation of a bursa which communicated with the joint. The bursa discharged through a fistula, and infection through the fistula gave rise to suppuration and to inflammation of the joint. Severe symptoms developed, with rigors and fever, and lymphangitis of the foot and leg. The joint was opened on the dorsal surface by a transverse incision, and the superficial suppuration by another incision on the dorsum of the foot. The clavus and the bursa were excised subsequently.

PHLEGMONE PROGREDIENS PUTRIDA

(*Putrefactive Phlegmon*)

Plate LXXIX, Fig. 101.

Pyogenic affections are especially dangerous when the infection is caused by very virulent bacteria, and also when bacteria invade a debilitated body (*e.g.* diabetes). In this case (Fig. 101), subcutaneous suppuration, following a slight wound of the great toe, rapidly spread to the tendon-sheath and the joint, necessitating amputation of the toe on account of the extensive infection and severe constitutional symptoms. Although the operation was made through tissues not yet inflamed, further suppuration occurred on the sole of the foot, which spread rapidly and destroyed the soft parts, tendons, muscles and fascia, and infected the metacarpal bones. The severity of the inflammation is shown by the great swelling around the metacarpus. This is not a case of the progressive suppuration which is common in diabetes, but one of secondary infection by bacteria of putrefaction, giving rise to a putrid, sanious inflammation. If pyogenic and putrefactive phlegmons are combined, there is not only rapid necrosis of all the tissues with extension of the process to the neighboring parts, but also general infection (*cf.* Fig. 108).

The appearance of the wound in this form of inflammation, which is also called gangrenous, is characteristic. Owing to the fibrinous exudation, the wound is coated with a diphtheroid membrane. This condition has been called "wound diphtheria"; but it is better to use the term diphtheroid, as cases of true infection of wounds with diphtheria bacilli are rare. In putrefactive phlegmon dry, unhealthy granu-

lations are present along with the diphtheroid membrane. There is also a sanious, fetid, dirty discharge from the wound, containing numerous pieces of necrosed tissue. Similar conditions are found in wounds in general infection.

In diabetics, these putrefactive phlegmons assume a very extensive and dangerous character, as the diabetic tissues constitute a favorable nutritive medium for bacteria, especially those of putrefaction, while the debilitated body offers little resistance to them. If an incision is made in these cases all the tissues are seen to be bathed in a dirty green fluid and in a state of necrosis, often consisting only of yellowish-green necrotic shreds. The skin, fascia, muscles and tendons are the first to be destroyed, while the bones resist longer. In our case, the pyogenic and putrefactive phlegmon had already loosened the periosteum from the bones and caused infection of the cortex and medullary cavity (osteomyelitis, cf. Fig. 104). The infection of the bones at first gave rise to severe rigors, but afterwards assumed a more chronic form of inflammation. There was also extensive lymphangitis and thrombo-phlebitis of the leg.

Treatment. In cases of putrefactive phlegmon, free incisions must be made in the diseased tissues as early as possible, as general infection often occurs rapidly from the action of toxins. If the process continues to extend in spite of the incisions, amputation through healthy tissues must not be delayed too long; otherwise the patient will succumb in spite of amputation.

In the phlegmonous inflammations occurring in diabetes, which often begin in the toes and spread destruction over the whole foot in a few hours, the conditions are especially complicated. If, after extensive incisions, the temperature does not immediately fall, amputation must be performed; otherwise general infection will occur rapidly. In any case of

phlegmonous inflammation in a diabetic patient death may occur from coma or heart failure.

In Fig. 101 there was a combination of pyogenic and putrefactive phlegmon of a progressive character in a diabetic patient. High temperature, rigors, dry tongue and somnolence suggested the commencement of general infection. Amputation was performed above the knee, owing to the presence of lymphangitis, thrombo-phlebitis in the leg, and also advanced arterio-sclerosis. The operation was performed under lumbar anæsthesia and led to healing.



Fig. 102. Phlegmone colli — Phlegmon ligneux.

PHLEGMONE COLLI (*Phlegmon of the neck*)
Plate LXXX, Fig. 102.

In the region of the neck, subcutaneous and sub-fascial phlegmons are common, owing to the numerous groups of lymphatic glands in this situation. Suppurative inflammation of these glands may be caused by affections of the mouth and pharynx, carious teeth, angina, otitis media, alveolar periostitis, foreign bodies, etc. Eczema and other affections of the head and face may also cause suppuration in the glands of the neck, especially in young individuals. The infection is generally due to staphylococci, sometimes streptococci and other bacteria. In lesions of the mouth and pharynx putrefactive bacteria are sometimes found in the buccal cavity.

Subcutaneous phlegmon in the neck manifests itself by redness of the skin, inflammatory infiltration and fever; later on fluctuation can be made out. In nearly all cases a circumscribed abscess forms on one side of the neck. Large abscesses may cause dyspnoea by pressure on the larynx, and dysphagia by pressure on the esophagus.

In the submaxillary region the inflammation occurs most commonly in the subcutaneous lymphatic glands, and the abscess is situated outside the capsule of the submaxillary gland. This must be distinguished from intracapsular suppuration of the submaxillary gland itself, which is called *Ludwig's angina* (*angina Ludovici*). In this case the symptoms are much more severe—fever, rigors, swelling in the buccal cavity and pharynx, causing difficulty in respiration and swallowing.

Infection of the sub-mental lymphatic glands gives

the sternomastoid muscle. Extensive cases require counter-incisions. The wounds should be drained by gauze tampons, as drainage tubes may injure the large vessels.

Wooden phlegmon of the neck sometimes requires multiple deep incisions, laterally and in the middle line.

Fig. 102 shows acute inflammation of the sub-maxillary lymphatic glands, with the formation of an abscess under the skin. It was treated under local anæsthesia by incision and drainage.



Fig. 103. Periostitis alveolaris purulenta — Parulis.

PERIOSTITIS ALVEOLARIS PURULENTA—PARULIS

(*Purulent alveolar Periostitis*)

Plate LXXXI, Fig. 103.

Parulis is a name given to purulent alveolar periostitis of the lower jaw, which usually gives rise to a subcutaneous abscess. It may be caused by lesions of the gums (*e.g.* after tooth-extraction with dirty instruments), fractures of the jaw, operations on the jaw, caries of the teeth, fistulas from the stumps of teeth. Infection of the periosteum of the alveolar portion of the lower jaw gives rise to a circumscribed subperiosteal accumulation of pus which descends to the submaxillary region and lies over the fascia covering the submaxillary gland. The signs of purulent inflammation are most apparent in this region, while symptoms at the seat of infection are often slight.

The symptoms commence with fetor of the breath, fever and rigors, and inflammatory infiltration in the submaxillary region. Soon afterwards the presence of fluctuation indicates abscess formation, after which the symptoms diminish. In most cases the suppuration is circumscribed, but sometimes there is diffuse inflammation, causing considerable infiltration of the soft parts and swelling and redness of the side of the face. There is then often trismus and œdema of the mucous membrane of the mouth, with difficulty in mastication and often difficulty in breathing. In these diffuse forms there are severe constitutional symptoms—rigors, fever, headache, etc.

Although the circumscribed form is harmless, the diffuse form may be dangerous to life, especially when improperly treated. Treatment of the circumscribed form by poultices may give rise to the diffuse form. If the pus is allowed to remain for long under

the periosteum, it may cause osteomyelitis of the jaw and all its consequences (Fig. 104). Meningitis and general infection may also occur from thrombophlebitis.

In the upper jaw, infection of the periosteum may also cause subperiosteal suppuration, which has not such favorable conditions for extension to the subcutaneous tissue as in the case of the lower jaw. Small abscess caused by morbid conditions of the teeth may burst into the mouth and cause no trouble, but more virulent infection may cause osteomyelitis of the upper maxilla, which rapidly extends over the whole of the bones of the face, and often causes death by general infection. In these cases there is infiltration of the upper part of the face, oedema of the eyelids, high temperature, rigors, headache, etc.

In these morbid conditions pyogenic inflammation generally staphylococcal, is often combined with putrefactive inflammation from bacteria in the mouth. We, therefore, find the fetid, dirty, reddish-brown pus, mixed with broken-down tissue, which is characteristic of putrefactive inflammation.

Differential Diagnosis. Although parulis of the lower jaw may cause swelling of the neck resembling glandular abscess, it can usually be distinguished by the history, and by inspection of the mouth. If the parulis has been present some time the bone becomes to a considerable extent denuded of its periosteum which distinguishes it from glandular abscess. In the upper jaw empyema of the antrum of *Highmore* may be mistaken for parulis, especially when the empyema has broken through the bony wall of the antrum and appears as an abscess under the gum. If the antrum of *Highmore* is translucent to light there is no pus in it; on the other hand, absence of translucency does not necessarily indicate the presence of pus, as this sometimes occurs in the normal condition.

Primary acute osteomyelitis commences with more severe symptoms—high fever, frequent rigors, etc.

Treatment. Circumscribed abscesses should be incised under local anæsthesia. Poultices are to be avoided. Diffuse inflammations should be incised under general anæsthesia before the formation of abscess. By this means the above-mentioned complications may be prevented. In parulis of the lower jaw an incision should be made through the skin and the pus evacuated by dressing forceps; in this way pus can be found which was not apparent from the external appearance. If the rough bone is found a large drainage tube should be inserted. The incision should be made about three-fourths inch below the border of the jaw to avoid the branches of the facial nerve which supply the muscles at the angle of the mouth.

In the upper jaw operation should be performed from the mouth; with the head hanging low, in cases of large accumulations of pus.

In all cases of parulis the teeth must be attended to; carious teeth and stumps, which have given rise to the condition, should be removed. Drains and tampons can be left out in a few days, when suppuration has ceased. If the movements of the jaw are limited, fluid diet may be necessary at first.

Fig. 103 shows a case of parulis arising from a carious premolar of the lower jaw. It began with pain and fever, and the formation of an abscess under the gum. Eventually, an abscess formed in the neck, after which the symptoms subsided. Under general anæsthesia an incision an inch long was made at the lower border of the abscess and fetid pus evacuated. Staphylococci and putrefactive bacteria were found in the pus. Owing to the previous treatment of the patient with poultices, the bone was considerably denuded of periosteum. Healing took place in fourteen days.

Osteomyelitis

OSTEOMYELITIS MAXILLÆ INFERIORIS

(*Osteomyelitis of the lower jaw*)

Plate LXXXII, Fig. 104.

OSTEOMYELITIS SCAPULÆ ACUTA

(*Acute osteomyelitis of the scapula*)

Plate LXXXIII, Fig. 105.

OSTEOMYELITIS HUMERI CHRONICA

(*Chronic osteomyelitis of the humerus*)

Plate LXXXIV, Fig. 106.

OSTEOMYELITIS TIBIÆ—NECROSIS TOTALIS

(*Osteomyelitis and necrosis of the tibia*)

Plate LXXXV, Fig. 107.

The term osteomyelitis is applied to pyogenic affections of bone in general, while in the stricter sense these are divided into purulent periostitis, osteitis and osteomyelitis. Since all three parts of the bone are generally the seat of suppuration and the process can only be localized clinically to the bones as a whole, and as the majority of cases begin with infection of the bone-marrow, the name osteomyelitis is rational.

Infection of the bones may result from lesions of the soft parts, compound fractures, operations (this was common after amputations in the pre-antiseptic days); after pyogenic affections of the neighboring parts (subcutaneous abscess, whitlow, otitis media). In the latter cases the periosteum is first infected, the cocci then invade the Haversian canals in the cortex and infect the medullary cavity. As in all pyogenic infections, the great majority of cases are caused by the *staphylococcus pyogenes aureus*; while the *staphylococcus albus*, *pneumococcus* and *streptococci* only in rare cases cause infection of bone.

Apart from the above-mentioned modes of infection this may take place through the blood; the medulla is then first infected, and the suppuration spreads to the cortex and periosteum, finally appearing as a subcutaneous abscess.

In all pyogenic affections (furunculosis, whitlow, quinsy, otitis media) the bone marrow is infected by staphylococci, but the power of resistance of the body is generally sufficient to withstand their action. The cocci remains harmless till the power of resistance of the body is weakened by some exciting cause, such as fracture, overexertion, exposure to cold, etc. Osteomyelitis may thus occur after injury to a bone, even after a slight contusion. In this case the resulting effusion of blood favors further growth of the cocci and leads to infection. It follows from this that, according to the circumstances, purulent infection of the bones may develop sometimes directly after and sometimes a long time after purulent inflammation in other organs of the body; also that, according to the number and virulence of the bacteria, it may assume an acute or chronic form, with corresponding violent or mild symptoms. Like all purulent inflammations, the process begins at the seat of infection with hyperæmia, exudation, suppuration, degeneration and regeneration; these processes assuming a special form corresponding to the structure of the bone. Thrombo-phlebitis may occur and give rise to metastatic infection by embolism in other parts of the body (bones, endocardium, meninges, etc.)

As the great majority of cases arise from blood infection, it is clear that the bones most liable to infection are those which are most richly supplied with blood-vessels, especially during their period of growth when they are most vascular. The diaphyses of the long bones are thus most often affected at their junction with the epiphyses. The lower ends of the femur and radius and tibia, and the upper ends of

the humerus and tibia are the places of predilection. Osteomyelitis is rare in the short bones and in the flat bones. It is also rare after the thirtieth year. According to the statistics of *Garrés*, in one-fifth of the cases several bones are affected simultaneously.

The symptoms of acute purulent osteomyelitis are more severe than in any other pyogenic affection. The deeper the infection, the greater is the virulence of the bacteria. Bacteria in the bone-marrow are under greater pressure than in any other tissue, and this increases their virulence. In young individuals osteomyelitis often occurs suddenly after an injury, with high fever, rigors, pains in the joints and severe constitutional disturbance. Pain on pressure and movement, and loss of function point to an affection of the bones. Serous effusion soon takes place in the nearest joint. Changes first appear under the skin when pus forms under the periosteum. The subperiosteal abscess appears as a sharply defined fluctuating swelling with hard borders, and the skin over it is tense and reddish blue. If the subperiosteal abscess bursts, it gives rise to intermuscular and subcutaneous infiltration, with redness and swelling of the skin, and oedema of the soft parts; the regional lymphatic glands are swollen and painful.

Although operation often only reveals a subperiosteal abscess, especially in children, in cases of hematogenous origin (blood infection) the cortex and medulla of the bone are also affected. Infection of the cortex is shown by the presence of yellow spots on the surface, which correspond to small holes discharging pus. After removal of the cortex, the infected medulla shows reddish-brown or yellowish spots, which may lead to the formation of a circumscribed abscess, or to diffuse suppuration in the medullary cavity. If the condition is not recognized early and the spread of infection arrested by operation, separation of the epiphyses or infection of the joint may occur, or general infection with death in

a few days. In extensive disease the whole bone is whitish-yellow; white from bloodlessness due to thrombo-phlebitis, and yellow from pus formation. Numerous pits are seen from which pus has been discharged under the periosteum.

The amount of necrosis corresponds to the degree and extent of infection. In subperiosteal necrosis the infected cortex and medulla may regenerate without loss of substance, especially when the pus has obtained an early exit. If the cortex has been for some time the seat of extensive purulent inflammation necrosis must result with the formation of a *sequestrum*. According to the extent of the inflammation this necrosis will be limited to one part of the bone or extend through the thickness and length of the bone partially or completely. In disease of the cortex the sequestrum is generally lamelliform, slightly corroded and pitted; in disease of the medullary cavity the sequestrum is, to a certain extent, a cast of the cavity, and in the form of a trough.

The sequestrum in osteomyelitis is large and continuous and may include the whole length and thickness of the diaphysis (Fig. 107), thus differing from the sequestra in tuberculous bone disease, which are generally multiple, small and much corroded. Such complete necrosis occurs in acute cases which have been operated upon too late and in chronic cases. The dead bone (sequestrum) becomes separated from the healthy bone by a zone of inflammatory demarcation, more or less rapidly according to its size.

In extensive necrosis the demarcation process may continue for months, so that patients who escape death from general infection may succumb from exhaustion, albuminuria or amyloid degeneration of the kidneys. Spontaneous expulsion of the dead bone should be assisted by operation (sequestrotomy).

The regenerative or osteoplastic process goes hand in hand with the degenerative. The purulent inflammation not only causes necrosis, but causes irritation which stimulates the periosteum to form new bone (osteoplastic periostitis). This results in thickening of the cortex at the seat of necrosis; and in cases of total necrosis, complete repair of the destroyed bone. This irregular formation of new bone is sometimes called the "sequestral capsule." There are numerous holes (cloacas) in this capsule where the periosteum has been destroyed. From these holes pus is discharged from the zone of inflammatory demarcation, and eventually the sequestrum, through a fistula in the skin (Fig. 107). The X-rays are useful in showing the extent of necrosis, and also separation of the epiphyses.

The whole process of degeneration and regeneration take much longer than in purulent inflammation of the soft parts, and the acute stage is followed by a chronic stage after the pus has been evacuated spontaneously or by operation. However, an acute relapse may occur at any time during the chronic stage, especially after improper treatment, or after an injury.

In distinction to this form of acute osteomyelitis there is a subacute form which is chronic from the beginning. In these cases there is often a history of previous acute inflammation of the bone, and the condition is really one of recurrence in a milder form, often at the age of puberty. Recurrence may also occur later in life, hence bones which have been previously affected with osteomyelitis must be regarded as places of less resistance and must be protected from the action of trauma and over-exertion.

The clinical symptoms in these cases often resemble rheumatic pains, but the pain is localized to one bone, or sometimes a definite part of a bone. There is often a history of pyogenic disease in youth, and scars and fistulas may be found in the bone con-

cerned or in other bones. The affected bone is often very tender to pressure at certain points. In the course of time the bone becomes thickened, and the diaphysis lengthened. The growth in thickness may be enormous at the seat of disease, the thickening being both periosteal and cortical.

The changes in the bone in chronic osteomyelitis are as follows: Sometimes there is a small sequestrum in the interior of the bone, shown as a clear spot surrounded by bony proliferation in an X-ray picture; sometimes a circumscribed abscess in the medullary cavity, shown by the X-rays as a round space surrounded by bone. If bony proliferation is absent the X-ray pictures resemble tumors or cysts in the bone. The diagnosis of chronic osteomyelitis is, therefore, sometimes difficult when there is no history or evidence of former osteomyelitis. Pain on pressure suggests the infective nature of the disease. In doubtful cases search may be made for staphylo-lysin, according to the method of *Bruck, Michaelis* and *Schultze*.

If large portions of the cortex and medulla are affected by chronic osteomyelitis large sequestra are formed, which seek a way to the surface in spite of the considerable formation of new bone. In these cases we find numerous cloacas in the bony capsule, subcutaneous abscess and fistulas (Fig. 106); while the whole bone is thickened, and the X-rays show changes in the periosteum, cortex and medulla.

A third form of chronic osteomyelitis is limited to the periosteum, under which a hyaline sero-mucoid fluid develops, forming a sharply defined, fluctuating swelling with hard borders. This has been called albuminous periostitis but is a form of osteomyelitis. Staphylococci are present in the fluid.

All these chronic forms are due to infection by less virulent staphylococci. However, every chronic osteomyelitis may become acute, especially when the bones are exposed to the effects of overexertion,

injury, or massage (performed on account of wrong diagnosis). Chronic fistulas in osteomyelitis may give rise to carcinoma (cf. Plate XIV). In the long bones both acute and chronic osteomyelitis may cause disturbance in growth, pseudarthrosis and contractures. Although the great majority of cases of acute and chronic osteomyelitis affect the long bones, both forms may occur in the short and flat bones; in the skull, after compound fractures, incised and punctured wounds; in the scapula, pelvic bones and vertebræ; in the bones of the face (after tooth extraction). In *Fröhner's* statistics, four hundred and seventy cases of osteomyelitis affected the long bones and thirty-four the short and flat bones. As the cortex is thin in these bones, there is greater destruction. Osteomyelitis of the cranial bones may spread through the diploë to half the skull, form large sequestra of the inner table, and epidural abscess. In the scapula the whole bone may be destroyed by multiple abscesses and sequestra, necessitating complete removal of the bone. In osteomyelitis of a facial bone, infection may spread to all the bones of the face, causing extensive destruction and consequent deformity. Osteomyelitis of the cranial and facial bones may give rise to meningitis.

In streptococcal osteomyelitis the pus is thinner and very abundant, and the disease is more severe like all streptococcal infections. In these cases the skin usually shows erysipelatous reddening.

Osteomyelitis after infection by typhoid bacilli or pneumococci can only be distinguished from the other forms by the history and by bacteriological examination.

Differential Diagnosis. Acute osteomyelitis may be mistaken for deep abscess, but this is made clear by incision. The redness of the skin in osteomyelitis resembling erysipelas is limited to the affected part and gradually diminishes. Acute osteo-

myelitis of the diaphyses is characterized by the severity of the symptoms, the marked swelling and the loss of power in the limb.

Chronic forms are most often mistaken for tuberculous bone disease, but the latter generally affects the epiphyses, while osteomyelitis attacks the diaphyses. Osteomyelitic fistula has hard borders and bright red granulations, and passes directly to the bone, while tuberculous fistula has yellow, slimy granulations, irregular borders and an irregular course through the deep parts (Figs. 125 and 130). In osteomyelitis the pus is reddish brown, in tuberculosis it is thin and greenish yellow. In doubtful cases an incision will decide the diagnosis; in osteomyelitis the periosteum and cortex will be found thickened and the sequestrum large and continuous; in tubercular bone disease there are multiple, small corroded sequestra.

Chronic osteomyelitis causing much swelling of the bone may be mistaken for syphilitic bone disease, especially in the tibia. In syphilitic bone disease the X-rays show a diffuse thickening of all layers of the bone, and a uniform dark shadow with irregular borders, corresponding to the periosteum; while, in osteomyelitis, dark shadows together with clear spaces are shown, corresponding to sequestra and abscesses respectively. If fistulas form in syphilitic bone disease they present the characteristic sharp borders and prolific granulation tissue round them (Fig. 122).

Osteitis deformans (*Paget's disease*) is characterized by affecting the whole extent of both tibias, and by the early appearance of marked curvature.

Osteomyelitic abscesses in the diaphysis, when they extend to the epiphyses may be mistaken for tuberculosis, but the pronounced new bone-formation is absent in the latter. Sarcoma and bone-cysts may also in some cases be difficult to distinguish from chronic osteomyelitic abscess, even by the X-rays.

In doubtful cases an exploratory incision may be made, or staphylolysin looked for.

In the majority of cases, however, the diagnosis of osteomyelitis is established by the history and the typical appearance, situation and course of the disease.

The earlier diagnosis is made and treatment commenced, the better the prognosis.

Treatment. In the most acute cases with purulent joint-effusion and signs of general infection (dry tongue, delirium, presence of bacteria in the blood) amputation is sometimes the only means of saving life.

In acute osteomyelitis incision must be made as soon as possible, before the abscess has broken into the subcutaneous tissue. After opening the abscess the bone must be examined; if it is unaltered it can be left alone. If the temperature does not fall after opening the abscess and the condition becomes worse, with rigors, etc., the bone must be laid open as far as the medullary cavity. This should be performed freely with a gouge; it is useless simply to bore holes as they do not give sufficient outlet for pus, nor for subsequent necrosed pieces of bone. On the other hand, in cases with severe constitutional symptoms, especially in children, the whole extent of bone should not be gouged at one sitting, owing to the severe shock, and the possibility of general infection; the gouging should be performed at several sittings. After gouging, the infiltrated bone-marrow must be scraped with the sharp spoon and the cavity drained with iodoform gauze. The wound must be kept open by a drainage tube to allow pus and sequestra to escape.

The after-treatment is sometimes hindered by narrowing of the opening in the bone from the formation of callus; if there is no suspicion of necrosis, this callus must be removed with the knife, to establish

sufficient communication with the medullary cavity. Complete immobilization is necessary in the extremities, to avoid spread of inflammation and the possibility of fracture.

Serous effusion into a joint must be punctured when extensive. Purulent effusion requires incision, and sometimes resection of the joint. If there is purulent arthritis with high fever and rigors, resection must not be delayed, or general infection may follow.

In chronic osteomyelitis it is best to wait till the sequestrum is complete and new bone has begun to form round it (X-ray examination) before performing sequestrotomy. If there are subcutaneous abscesses these must be opened. As small sequestra and abscesses often cause considerable pain, in some cases the bone must be gouged when the X-ray examination shows no changes. The operation is troublesome, as the small sequestrum or abscess is often situated in the middle of hardened sclerotic bone. The fistulas in chronic osteomyelitis must be freely opened up and the callus removed. The cavity in the bone left after gouging must be left open and drained till healing takes place from the bottom. Immediate plugging of the bone cavity with iodoform is only of use in a few cases of circumscribed chronic osteomyelitis, as in extensive cases the plugs are often expelled through a fistula; but when the cavity is filled with fresh granulations, all cases of osteomyelitis can quickly be made to heal with plugging. The cavity is then scraped, disinfected with peroxide lotion, dried with *Hollander's* hot air apparatus, and filled with a mixture of iodoform, glycerin and spermaceti. Whenever possible, the periosteum should be united over the plug and a covering of skin made over the cavity. Strict asepsis is necessary.

Frequent recurrences in chronic osteomyelitis, with emaciation, albuminuria, etc., necessitate amputation. Contractures must be treated by extension on a splint, or when they cannot be extended, by resection.

Large defects in the skin can be covered by pedunculated flaps.

In the flat bones subperiosteal removal of the whole bone is often necessary (*e.g.* scapula). This may be followed by complete regeneration and restoration of function. In osteomyelitis of the cranium sequestra and epidural abscesses must be evacuated through a large trephine hole, which can afterwards be repaired by bone grafting.

Treatment of acute osteomyelitis by passive hyperæmia is to be condemned, as it obscures the signs and symptoms. It may also lead to diffuse suppuration by thrombo-phlebitis, rendering amputation necessary; but its chief danger is general infection.



Fig. 104. Osteomyelitis maxillae inferioris.

OSTEOMYELITIS MAXILLÆ INFERIORIS

(*Osteomyelitis of the Lower Jaw*)

Plate LXXXII, Fig. 104.

This figure shows chronic osteomyelitis in a girl of nineteen, which occurred in connection with tooth extraction. Osteomyelitis of the lower jaw often occurs after tooth extraction, when there is much inflammation of the gum and periosteum, or when the alveolus is extensively injured. It may also follow injuries to the jaw. Osteomyelitis of the lower jaw, due to blood infection, is generally combined with disease of other bones, and occurs especially in children. Acute osteomyelitis of the lower jaw commences with high fever, rigors, œdema of the face and mucous membrane of the mouth, difficulty in breathing and swallowing, headache and delirium. It is often fatal from meningitis or general infection. In some cases the whole of the lower jaw may become necrosed.

In the chronic form (Fig. 104) a painless circumscribed or diffuse painless swelling slowly develops in the lower jaw. The skin gradually becomes tense, red and œdematous; one or more fistulas develop, and later on necrosis takes place. In extensive cases the teeth become loosened and trismus may occur. In the stage of painless swelling the case may resemble cystic adenoma. In actinomycosis the swelling is situated in the floor of the mouth and in the muscles, and only extends to the bones later on.

Osteomyelitis of the lower jaw should be treated by early incision down to the bone, at the lower border of the jaw. Healing without necrosis occurs more often than in the long bones. If necrosis occurs it is best to wait, in chronic cases, till sufficient

new bone is formed, so as to avoid fracture of the jaw during removal of the sequestrum.

Sequestra are best removed by external incisions. The cavity should be plugged with iodoform gauze for a long time. In extensive necrosis, bone grafting may be tried, or the patient may wear a prosthesis.

In Fig. 104 the fistula was opened up; after which the discharge diminished, but the swelling of the bone remained and the fistula did not heal, indicating necrosis. The X-rays showed diffuse swelling of the jaw.

A condition affecting the bones, observed by *Billroth* in workers in mother-of-pearl, which resembles osteomyelitis, and chiefly affects the lower jaw, undergoes spontaneous resolution; so long as the patients are not exposed to fresh injury through their work.

Phosphorous necrosis of the lower jaw, which occurs in workers in yellow phosphorus, is probably due to infection of the bone. The phosphorous vapor causes ulceration of the gums, through which the periosteum and bone are infected. This condition gives rise to great swelling of the whole of the lower jaw. The teeth become loose and fall out. The gums become ulcerated and fetid, so that many patients succumb to septic pneumonia or to general septic infection. The bone becomes both sclerosed and brittle. After some years total necrosis occurs with a row of fistulas along the lower border of the jaw.

As there is generally total necrosis in these cases, partial resection is useless, and subperiosteal resection of one or both sides of the jaw should be performed. After this regeneration of the jaw takes place if the periosteum has been preserved, and relapses are avoided.

Phosphorous necrosis (which is fatal in fifty per cent. of the cases) has been prevented by the prohibition of the use of the dangerous yellow phosphorus in the manufacture of matches.



Fig. 105. Osteomyelitis scapulae acuta.

OSTEOMYELITIS SCAPULÆ ACUTA

(*Acute Osteomyelitis of the Scapula*)

Plate LXXXIII, Fig. 105.

This is a case of acute osteomyelitis of the scapula following an injury. A few days after the injury a swelling appeared over the whole scapular region as far as the supra-clavicular fossa, accompanied by fever and rigors. The skin became red and mottled, and a large fluctuating subcutaneous abscess developed. The function of the shoulder-joint was abolished. An incision was made and pus evacuated; the bone at the seat of injury was infiltrated with pus. Healing took place without any necrosis.

In osteomyelitis of the scapula, especially when due to blood infection, an abscess usually forms at the anterior border of the scapula, as the osteomyelitic focus in this mode of infection is situated in the body of the bone. The pus is at first limited by the subscapularis muscle; on the other hand, the pressure of the muscle causes rapid extension of suppuration in the medulla of the bone. The abscess may thus not be recognized till it breaks through into the axilla. An early symptom of osteomyelitis of the scapula is painful effusion into the shoulder joint; on this account it may be mistaken for an affection of that joint, the true seat of disease only being revealed after incision. In doubtful cases the anterior surface of the scapula should be exposed by an incision in the axilla. In most cases of osteomyelitis of the scapula, the wound does not heal after incision of the abscess; the occurrence of multiple abscesses and necrosis is unavoidable, owing to the extension of suppuration through the medulla of the bone. For

this reason the disease may last for years. In these cases, and also in acute cases where incision shows extensive destruction of the bone, subperiosteal total extirpation of the scapula is indicated, taking care to preserve the muscular attachments and the important nerves. This is especially indicated in acute osteomyelitis of the flat bones, which often gives rise to early general infection. After total extirpation of the scapula relapses are avoided, and complete regeneration of bone with normal function is possible (*Bockenheimer*).

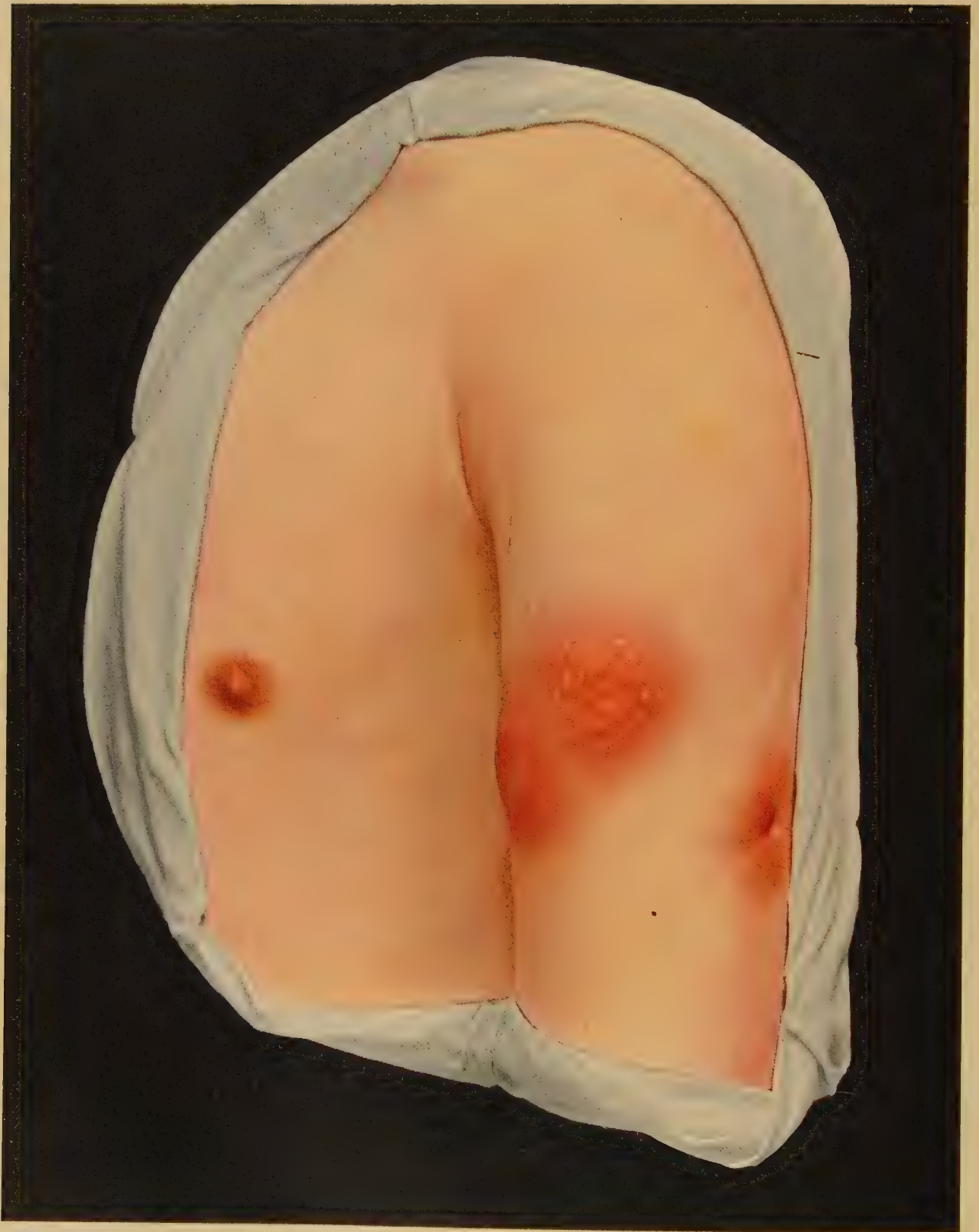


Fig. 106. Osteomyelitis humeri chronica.

OSTEOMYELITIS HUMERI CHRONICA

(*Chronic osteomyelitis of the Humerus*)

Plate LXXXIV, Fig. 106.

Fig. 106 shows a painful club-shaped swelling of the left humerus, which gradually developed at the age of puberty, in a patient who had frequently suffered from tonsilitis in childhood. The patient attributed it to over-exertion at his work as a blacksmith. A year after the onset, a fistula formed at the posterior and external side of the arm, with hard borders and red granulations at its orifice. A probe passed down the fistula discovered rough bone, denuded of periosteum. Subcutaneous abscesses formed at the front of the arm, where the skin was thin and reddened. Examination by the X-rays showed a sequestrum, along with new bone formation. Chronic osteomyelitis of the diaphysis of the humerus was diagnosed. An incision was made down to the bone in the lower third of the outer side of the arm, avoiding the radial nerve. The periosteum was destroyed at one place and a cloaca was found leading to a sequestrum. The sequestrum was removed by carefully gouging the bone; the cavity was scraped and plugged, and the fistulous track with its hardened walls excised. The subcutaneous abscesses were opened and scraped. The arm was immobilized for a long time. Healing took place after some months, and the patient was told to choose a lighter occupation in order to avoid recurrence of the disease.

OSTEOMYELITIS TIBIÆ—NECROSIS TOTALIS

(*Acute Osteomyelitis and Necrosis of the Tibia*)
Plate LXXXV, Fig. 107.

In this case acute osteomyelitis of the tibia in a child, aged nine years, commenced with severe pain in the leg and knee joint, accompanied by high fever and rigors. There was no history of a previous attack. A few days before the onset the child received a blow on the tibia. In spite of the severe clinical symptoms and the marked swelling of the knee joint, operative treatment had been neglected, and, only when a subcutaneous abscess developed, was an incision made. Although the acute symptoms gradually subsided after this, the swelling of the leg remained, and the wound discharged fetid pus. In a few months almost the whole shaft of the tibia became necrosed. Fig. 107 shows the yellow necrosed bone, with the open medullary cavity containing slimy granulations. Between the necrosed bone and the healthy bone are granulation tissue and pus. As the leg had not been properly fixed, a fracture occurred at the lower part of the tibia. The condition of the child on admission to hospital was very bad, owing to the prolonged suppuration. Examination by the X-rays showed that the sequestrum extended further down, and that a thick, bony capsule had already formed behind and at the sides.

Under an anæsthetic, the wound was extended downwards, the necrosed bone removed, the cavity scraped and plugged, and the leg put up on a splint with extension, to correct the position of flexion. The equinus position of the foot, due to insufficient fixation, was gradually corrected.

Such extensive necrosis could have been avoided by early gouging of the bone and proper after-treatment.



Fig. 107. Osteomyelitis tibiae — Necrosis totalis.

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